THE AMERICAN JOURNAL OF ROENTGENOLOGY AND RADIUM THERAPY

VOL. XXVIII OCTOBER, 1932 No. 4

DISSECTING ANEURYSM OF THE AORTA

WITH SPECIAL REFERENCE TO ITS ROENTGENOGRAPHIC FEATURES*

By FRANCIS CLARK WOOD, M.D., EUGENE P. PENDERGRASS, M.D., *and* HERMAN W. OSTRUM, M.D.

PHILADELPHIA, PENNSYLVANIA

INTRODUCTION

ABOUT 400 cases of dissecting aneurysm of the aorta have been reported. A correct ante-mortem diagnosis, confirmed at necropsy has been made in 6 cases.[5,22,28,33,34,56]† In two of these,[5,22] the roentgenographic findings played an important part in the diagnosis.

In the majority of instances, the patient has survived the formation of a dissecting aneurysm for a considerable period of time, and in about 10 per cent of reported cases, rupture of the aneurysm back into the aortic lumen has been followed by "healing." Accurate diagnosis and careful management might prolong the lives of some patients, and possibly increase the percentage of "recoveries."

This paper consists of a review of the subject of dissecting aneurysm and a report of 11 new cases (with 6 from the literature). Special attention has been given to the diagnostic features. All cases reported in the literature, in which roentgenographic observations have been made[5,17,19,22,41,52] have been briefly reviewed. Roentgenograms of 3 of these cases[5,22,52] have been obtained from the authors and are reproduced (Figs. 2, 3 and 4). Four new cases with roentgenographic study and necropsy are reported (Cases 1, VIII, IX and X; Figs. 1, 5, 6 and 7), and an analysis of the roentgenological features of dissecting aneurysm has been made.

DEFINITION

By the term "dissecting aneurysm" is meant the development of a circulation pathway between the layers of the vessel wall in which blood circulates.[29]

HISTORICAL ASPECTS

Morgagni[35] was probably the first to describe a case of dissecting aneurysm. The patient was a fat woman, aged fifty, "of a sober disposition, and naturally rather inclined to sadness and taciturnity." She was "subject to no disease" . . . and having her menopause. One morning, when getting ready to go to work she "cried out 'Oh' and spoke not another word, but died instantly as she leaned with her arms on the beam." At necropsy it was found that "the blood had by degrees made itself a way through one of the intervals of this kind (ulcerations in the aortic intima), and had come out under the external coat of the artery; and then by raising it, as a large kind of ecchymosis . . . and finally, by distending

* From the Robinette Foundation, and from the Departments of Radiology of the Hospital of the University of Pennsylvania and the Philadelphia General Hospital.

† Osler[38] and Finny[9] have been mentioned in the literature as suspecting the diagnosis of dissecting aneurysm in their respective cases. This is not borne out by their own statements.

it more and more, had burst through this external coat in one place, and had poured itself out within the pericardium."

Although Morgagni, Allan Burns and Maunoir knew and described this pathological entity, Laennec is usually credited with giving to it its present name of "aneurysma dissecans."[29]

The best review of the entire subject is probably that by Peacock,[42] who has made a careful analysis of its various features on the basis of a collected group of 80 cases.

ETIOLOGY

There are two factors which are probably essential to the production of a dissecting aneurysm. The first is a *sudden rise of blood pressure*, usually dependent upon some trauma or strain. A good state of cardiac compensation is probably necessary to bring this about. In many cases, the initial intimal rupture has occurred during strenuous exercise, but milder types of exertion incident to sitting up in bed, mounting a horse, walking, vomiting or defecating, have been held responsible for the accident in certain instances. The second necessary factor is some *abnormality in the vessel wall.* Paschkis[40] has reported a case of "aortic rupture in the presence of an intact vessel wall," but his case report belies the title, in that a thinned-out area, which looked like a small scar, was found near the site of rupture. Lampert and Müller,[27] and Schmidt[48] have shown that a normal aorta will not give way unless subjected to an intravascular tension (900 to 1,500 mm. Hg) which markedly exceeds a level likely to be reached during life.

The morphological abnormalities in the vessel wall which seem to be necessary are two-fold. The first is a *fragile intima* which can be ruptured by a sudden rise of blood pressure. However, intimal rupture can occur without producing a dissecting aneurysm.[3,29,30,39] One must probably, therefore, postulate a second factor, namely, a *media which is readily dissectable.* Babes and Mironescu[2] believe that a pre-existing dissecting mesaortitis is responsible for this separability of the various layers of the aortic wall, and is the primary underlying cause of dissecting aneurysm. Whitman and Stein[55] in this connection report an interesting case of dissecting mesaortitis without dissecting aneurysm.

The specific cause for these morphological changes in the vessel wall which predispose to dissecting aneurysm is not known. *Chronic alcoholism* has been mentioned in the past history of many cases, but direct evidence in support of its etiological relationship to dissecting aneurysm is lacking. *Syphilis* has been considered by some authors[39] to be one of the causes. It is true that many patients with dissecting aneurysm have had positive blood Wassermann reactions, and have shown syphilitic aortic lesions at necropsy. However, in the majority of cases there has been no evidence of syphilis. Some observers[14,29] believe that syphilitic mesaortitis, by binding together the layers of the aortic wall, inhibits the formation of a dissecting aneurysm. In our series of 11 cases, there were 2 (Cases VIII and X) with positive blood Wassermann reactions and 2 others (Cases I and XII) with negative serology which showed typical histological syphilitic aortic lesions. Apparently a dissecting aneurysm can form, either in the presence or in the absence of aortic syphilis. Syphilitic aortitis is probably not a predisposing cause of dissecting aneurysm. However, we doubt whether it plays any definite part in preventing the formation of such a lesion. *Congenital weakness of the aortic walls* has been suggested as an etiological factor,[19,49] and may well be in certain instances. *Age* may play some part. MacCallum[31] states that it is very difficult to separate the various layers of a normal youthful aorta, whereas in an old, sclerotic vessel this separation can be accomplished with relative ease. *Hypertension*, whether it be a local type due to coarctation of the aorta,[18,49] or a general one, such as that seen in hypertensive cardiovascular disease, has frequently been a forerunner of dissecting aneurysm. *Arteriosclerosis* has

been present almost invariably in the reported cases.

Dissecting aneurysm has been produced experimentally in dogs by three different observers.[8,10,26] In each instance it occurred incident to an investigation of the arteriosclerosis caused by repeated injections of epinephrine (0.1 to 0.6 mg. was injected every second day for a period of about two months). At necropsy marked disease of the aortic walls was found. The intima showed atheromatous degeneration, and the media pronounced disruption of the elastic fibers. These observations lend weight to the belief, long entertained by clinicians, that arteriosclerosis and hypertension are the two most important predisposing antecedents of dissecting aneurysm.

PATHOLOGICAL ANATOMY

Dissecting aneurysm occurs, as a rule, in the aorta or in one of its main branches, but it has occurred in other arteries (pulmonary,[37] cerebral, splenic[29] and thyroid[25]).

(*a*) *The original tear in the intima* is located most frequently near the semilunar valves,* but it may be situated in any part of the aorta. Peacock[42] states that when it occurs near the aortic valve it is generally transverse in direction, whereas when located in the descending aorta, it is usually vertical. It may take place at the site of an atheromatous ulcer, but necropsy evidence does not establish this as an invariable occurrence by any means. Often the sharpness of its edges makes it resemble a knife cut (Case XVI).

(*b*) *The Dissection.* In the past, the blood was considered capable of dissecting its way between any of the various layers in the aortic wall, but this is probably not the case. Since Pennock's[43] and Goddard's[16] original observations, the dissection in every carefully studied instance has occurred within the substance of the media, usually between the middle and outer thirds (Case I). Babes and Mironescu believe that this is due to the fact that the dissecting mesaortitis, which they consider to be the invariable forerunner of dissecting aneurysm, affects primarily this part of the media. The circular extent of the dissection varies markedly but it usually involves one-half to two-thirds of the aortic circumference. Occasionally there is a complete separation of the inner from the outer layers, so that there is a "a sac within a sac."[3,43,54] The distance which the dissection may travel up or down the aorta also varies. If the primary rupture is near the aortic valve, the length of the dissection may be curtailed by an early death due to perforation of the aneurysm into the pericardium. (Case XV; exception—Case XVI.) On the other hand, if the intimal tear occurs in the arch or in the descending aorta, the dissection may extend to the iliac[54] (Cases IX and XIII), femoral[1,22] (Case XVI), and even the popliteal arteries.[29] During the dissection, some of the branches of the aorta may be torn off, their connection with the lumen of the aorta be severed, and their blood content thenceforth be derived from the aneurysmal cavity itself. Often they are partly occluded by external pressure, since the dissection frequently extends out along them.* Sometimes they are completely obstructed and thrombosed. Localized dilatations or sacculations of the outer wall of the aneurysm are frequently seen. Although these dilatations may occur at any point along the course of the aorta, they seem to be particularly prone to involve the distal portion of the aortic arch and the first part of the descending aorta (Cases III, IV, VI, IX, XII, XIV and XVI). The importance of this from the roentgenographic standpoint will be discussed later. The frequency of this location may be dependent upon increased internal pressure at this point in the arch.

(*c*) *The End-Results.* In most instances death occurs as a direct result of the dissecting aneurysm itself. Occasionally the patient dies from cardiac decompensation (Cases V and X), intercurrent infection

* Lundberg[30] explains the frequency of this location.

* Crowell[4] shows, in a table, the frequency with which the various branches of the aorta are involved in the dissection.

(Case XI) or an unrelated circulatory accident, but, as a rule, *external rupture* causes death. In fully half of all cases the external rupture takes place into the pericardial sac.[4]* Perforation has occurred into the right auricle,[29] pulmonary artery,[33] pleural cavity[24] (Case IX), mediastinum[36] (Case I), retroperitoneal connective tissue (Case I), esophagus, and lungs.[29]

In a smaller number of cases *internal rupture* occurs; that is, the dissecting blood reënters the aortic lumen through a weak spot in the intima, or at the site of avulsion of one of the large branches of the aorta.† This does not preclude the subsequent occurrence of an external perforation. However, when the internal perforation is large, and the blood entering the aneurysmal sac from above is able to leave it from below with sufficient rapidity to reduce the pressure in the aneurysm, "healing" may take place.‡ Subsequently the aneurysm may be entirely lined by newly formed intima and elastica (Cases V, X and XII), and the patient may live for many years.[38] The new inner lining of the aneurysmal sac is subject to pathological change and may take on a close resemblance to the original aortic intima in the course of time. This led some of the earlier observers of healed dissecting aneurysm to the erroneous conclusion that they were dealing with a congenital anomaly of the aorta ("double-barreled aorta").

Bostroem discussed the factors which may be responsible for bringing about internal rupture and subsequent healing of a dissecting aneurysm. He investigated the question from the standpoint of age, sex, height of blood pressure, etc., but came to no very definite conclusions. He suggested that the location of the initial tear in the intima may exert a certain amount of influence upon the outcome, in that a primary perforation in the arch or descending aorta is less apt to lead to early rupture into the pericardium than one situated just above the aortic valve, and may, therefore, offer a greater opportunity for recovery. Moreover, he stated that the possibility of reëntrance of the dissecting blood into the aortic lumen may be greater if the primary intimal tear is large, if the dissection proceeds rapidly, and if the blood pressure in the aneurysm is sufficiently high to produce compression and emptying of the normal aortic channel.

One other process should be mentioned by which "healing" of a dissecting aneurysm may take place, namely, clotting and subsequent organization of the extravasated blood. Its preliminary stages have been observed by Adami but complete healing on this basis has never been reported.

The effect of healing of a dissecting aneurysm might be likened to the effect of healing of a cardiac infarct. Death may be delayed, occasionally for years; the circulation is more or less crippled by the lesion, and the underlying pathological process which caused the lesion is still present and usually progressing.

Cardiac hypertrophy is usually present in patients with dissecting aneurysm. It may be due in part to the hypertension so frequently present in these individuals. Occasionally in healed cases, it may be produced by the dissecting aneurysm itself through the obstruction which this lesion offers to the blood current in the aorta (Cases V and X).

SIGNS AND SYMPTOMS

Crowell stated that the ages of reported cases of dissecting aneurysm have ranged from fourteen to ninety-five. Hall reported

* One hundred and forty-six of Bostroem's collected series of 177 cases died by external rupture; 90 of the 146 by rupture into the pericardium.[3]

† Forty-eight of Frei's[12] 273 collected cases; 20 per cent of Crowell's[4] collected cases.

‡ Seven of Peacock's collected series of 80 cases were "healed" (1863). Seventeen of Bostroem's collected series of 127 cases were "healed" (1888). Adami[1] collected 39 instances of "healed" or healing dissecting aneurysm (1896) and gave an interesting summary of the main facts about each in a bibliographic table. Although we have not made a complete search for all the "healed" cases reported since Adami's time, we have encountered records of 13 additional ones. (Goodman, Hall, Loeschke, Osler and McCrae, Hubschmann, Hart, Schede, Schilling, von Schnurbein (2 cases), Dumas et al, and Cases X and XII). It appears that about 10 per cent of the cases of dissecting aneurysm reported have been "healed" cases. Probably the actual percentage of cases which undergo "healing" is smaller than statistics compiled from the literature would seem to indicate, because of the added incentive to report the rarer "healed" cases.

a case in a boy aged seventeen; one of our patients (Case x) was twenty-eight years of age. Eighty per cent of patients, however, have been over forty years of age. Males are more frequently affected than females, the ratio being 1.64 to 1.[4] Adami pointed out that this preponderance in males is much less than that pertaining in saccular aneurysm.

Onset. The impression is obtained from the literature that the formation of a dissecting aneurysm is invariably a painful process. However, in a certain number of cases the history of the patient contains no mention of a severe attack of any kind, in spite of the finding at necropsy of a dissecting aneurysm[3,20,41,53] (Cases I, VI, VIII, X, XII and XIII). Four possible explanations for this have occurred to us. In the first place, such an attack may have taken place but not have been recounted by the patient either because he was more or less insensible at the time of the accident (Case X), or because it had happened so long before that he had forgotten it (Case XII), or because the physician failed to question him specifically.* In the second place, the pain incident to the formation of a dissecting aneurysm may be dependent upon adventitial distention rather than upon medial dissection.[50] If, therefore, one could conceive of a case in which the dissection took place by displacing the intima inward, rather than by distending the adventitia, and in which early reëntrance of the dissecting blood into the aortic lumen occurred, one could explain the absence of severe symptoms in occasional cases on this basis. In the third place, Sutton and Lueth have obtained evidence which throws some doubt upon the previously accepted belief that aortic adventitial distention produces pain. They believe that aortic distention produces dyspnea but that pain does not appear until hemorrhage into the surrounding tissues has occurred. In the fourth place, the formation of a dissecting aneurysm may be a slow process in certain cases. There may be a very gradual seepage of blood between the layers of the media, rather than a sudden rapid influx with violent adventitial distention. It would seem reasonable to suspect that such a process might be relatively painless.

The symptoms which usually mark the onset are very striking. The patient is seized, during excitement or sudden exertion, with terrific tearing, rending pain, the location of which is probably dependent to some extent upon the location of the original intimal perforation. It is usually, therefore, in the precordium or upper part of the chest, but may be in the back or abdomen.[20] As the dissection migrates down the aorta, the pain may follow its course and be referred successively to the shoulders, arms, back, lower spine, abdomen and legs (Case IX). Latham and Swaine described the onset in their patient as follows: "There was a sudden and violent pain, as though the chest were being torn open from side to side by some outward force. It was instantly followed by a second agonizing pain which seemed to dart from the midsternum down the left of the spinal column, and only to be arrested a few finger's breadth below and to the left of the umbilicus, at which point the patient thought he could hear a distinct crack." However, such a definite migration of pain is by no means a constant feature, and may be entirely absent. The pain is continuous and is accompanied as a rule by shock; restlessness, drop in blood pressure, sweating; and sometimes by vomiting.

In a certain proportion of cases, death follows promptly due to external perforation of the aneurysm, usually into the pericardial sac[35] (Case XV). In the majority of instances, however, there is a period of varying length* intervening between the initial symptoms and the final exitus, during which it is possible to diagnose the pathological lesion in certain cases. The fact that this has rarely been done is probably dependent, not so much upon the

* In a considerable number of patients, cerebral disturbances incident to hypertensive cardiovascular disease entirely mask the symptoms of dissecting aneurysm.

* Hours, days, weeks, and occasionally months or years.

extreme difficulty of the diagnosis, as upon the rarity of the lesion. It is not as a rule considered in the differential diagnosis, and the signs and symptoms are so little known to the average physician that an intelligent elicitation of them is rarely undertaken.*

The following are signs and symptoms of diagnostic importance which may be elicited during the interval between onset and death:

1. The pain, as a rule, partly or even completely disappears.

2. Hissing and rumbling murmurs are occasionally heard over some part of the aorta.[5,14,28]

3. An aortic diastolic murmur may appear. Several cases of dissecting aneurysm,[45] and of aortic intimal rupture without dissecting aneurysm[30] have been reported, in which a definite aortic diastolic murmur was present, but in which no evidence of aortic valvular disease was found at necropsy. This phenomenon was present in 3 of our cases (Cases I, V and X). If the intimal rupture is in the ascending aorta, Lundberg's explanation seems valid, namely, that the valve cusp directly below the rupture is displaced downward into the heart by the intraaortic pressure, causing imperfect valve closure. In Case I, however, there was no intimal rupture in the ascending aorta. It seems reasonable to suppose that the maladjustment of the valve leaflets in this case may have been due to the dissecting blood mass which extended back through the media to the aortic valve. The mechanism of the production of aortic insufficiency in this case, therefore, might have been similar to that described by Lundberg.

4. In many cases there is a definite anemia (Cases I, VII, VIII and IX) probably due, at least in part, to escape of blood into the aneurysm, mediastinal tissues, pleural cavities, etc.

5. Fever (Case IV) and leucocytosis (Cases I, VII, VIII, IX and XIII) are usually present, due to absorption of products of tissue break down in the region of the aneurysm.

6. Hemoptysis (Case X), hematemesis (Case XIII) and melena may occur.

7. A very important group of phenomena appears as a result of the partial obstruction, complete obliteration, or severance, by the aneurysm, of certain of the large branches of the aorta. (*a*) The pulse may be weak or absent, and the blood pressure correspondingly reduced in one or more of the arteries in the neck, arm or leg.[6,20,56 (and Case IX)] (*b*) The area whose blood supply is interfered with by this process may be cold,[6] pallid and painful (Case IX) and may even become gangrenous. (*c*) A cerebral ischemia may give rise to dizziness, unconsciousness, or other symptoms of disturbed central nervous system function[44]* (Cases IV and X). (*d*) Symptoms of disturbed spinal cord function have been reported in a number of cases,[4,6,11,13,28,31,41,54] and have been attributed to interference with its circulation by obstruction or avulsion of a number of intercostal arteries. Paralysis of one or both lower extremities, and loss of sphincter control of the bladder and rectum are the most common resultant phenomena. Erb describes a somewhat analogous symptom-complex in the dog in which he produced a dissecting aneurysm by experimental means.

8. Venous obstruction by the aneurysmal mass sometimes produces dilatation of the brachial, cervical or thoracic veins (Cases I and X). Occasionally it may produce edema.[56]

9. Pressure on the esophagus or trachea may cause dysphagia[41] or cough.

10. Mechanical pressure exerted by the mass on the celiac and other retroperitoneal sympathetic ganglia is supposed to be responsible for abdominal pain and distention in certain cases.[14,31 (and Case I)]

11. Signs of fluid in the pleural cavities are frequently elicited. They usually appear

* None of our cases were diagnosed ante mortem.

* In many cases one cannot be sure whether these phenomena are directly due to the aneurysm or to concomitant cerebral arteriosclerotic changes.

on the left side, probably because the lesion which produces the fluid is usually on the left. In the one case in which the lesion was on the right (Case x) the pleural collection was on the right. The fluid may be an effusion (Cases iv and vii), dependent either upon circulatory failure, upon blocking by the aneurysmal mass of veins or lymphatics draining the pleura, or upon irritation of the pleura by subserous hemorrhagic infiltration. In certain cases (Cases i, ii, iii, iv and ix) the fluid may be partially or completely bloody, due to a nonfatal rupture of the aneurysm into the left pleural cavity.

12. Transient hemoglobinuria has been considered by Hanser to be a finding of potential diagnostic value in certain instances. He reports a case in which this phenomenon occurred and outlines the mechanism by which it may have been produced, as follows: The aneurysmal mass blocks the thoracic duct and other mediastinal pathways by which lymph is ordinarily transported upward from the abdominal cavity and retroperitoneal tissues. This causes an increase in pressure in, and a dilatation of, the abdominal and retroperitoneal lymphatics. Those in the neighborhood of the renal pelvis partake in this dilatation, and allow some of their contents to escape into the urinary passages. Hematogenous products derived from the degenerating blood in the aneurysm are contained in these dilated lymphatics, and therefore appear in the urine. At first glance, this appears to be a rather nebulous hypothesis, but it is based upon an apparently careful and intelligent necropsy study which showed dilated retroperitoneal lymphatics filled with a reddish-colored fluid, and a group of distended lymph vessels in the renal pelvis containing a similar material. The fact that these hematogenous products were not present in the glomeruli or renal tubules was considered to be evidence against their transmission through the kidney itself. No other case report in our series (except possibly Case xiii), or in the group which we have reviewed from the literature, mentions the occurrence of hemoglobinuria, but since it is said to be an early and transient phenomenon, it may have been overlooked. It is not our intention to pass judgment upon the merits of the presence of hematogenous products in the urine as a diagnostic sign in dissecting aneurysm, but to call attention to it as a relatively unknown and possibly important phenomenon worthy of further investigation. In certain instances it might give rise to the mistaken impression that the pain caused by the dissecting aneurysm was due to renal calculus.

13. Occasionally some part of the dissecting aneurysm itself can be seen or palpated.[14,28] Since, however, the major portion of the lesion is usually confined within the thorax, the mass produced by it is not generally detectable by the unaided senses. Occasionally dissection along one of the carotid vessels may produce a palpable mass in the neck.

14. The diagnostic possibilities afforded by roentgenography in cases of dissecting aneurysm were brought to our attention in 1927 by the findings in a patient in the Hospital of the University of Pennsylvania (Case i). The film of this patient's chest presented a most unusual appearance (Fig. 1), the significance of which was not appreciated until necropsy. A review of the literature reveals that roentgenographic observations in cases of dissecting aneurysm have been reported by Goodman, Davy and Gates, Hirschboeck and Boman, Hall, Patrick and Taylor, and Tidmarsh. Through the kindness of these authors we have been able to collect a good many data on their cases, including photographs of three (Figs. 2, 3 and 4). Three other cases with roentgenographic films and necropsy reports have been found among the records of the Philadelphia General Hospital (Cases viii, ix and x; Figs. 5, 6 and 7). The roentgenographic appearance of the dissecting aneurysms in these 7 cases varies considerably, depending upon the location and pathological anatomy of the lesions. More information can

FIG. 1. Case I. The left chest is densely clouded by an effusion. The mass of the dissecting aneurysm can be seen forming an arcuate excrescence in the region of the aortic knob. There is a smooth edged, convexly curved shadow along the innominate artery due to an extension of the dissection along this vessel. The heart and trachea are displaced to the right. The combination of features present in this roentgenogram should suggest the diagnosis of dissecting aneurysm to the roentgenologist.

therefore be derived from inspection of the plates themselves than from a verbal description of them. Nevertheless a brief summary of the main features might be of value.

(*a*) There is usually a deformity of the supracardiac shadow. Sometimes this consists of a mere widening (Case VIII, Fig. 5), but often an arcuate excrescence is seen arising from some part of the arch (Cases III, VII and X; Figs. 2, 4 and 7), or extending to the left from the first part of the descending aorta (Case IV, Fig. 3; Case VI; and Case IX, Fig. 6). Such shadows are due to localized sacculations in the aneurysm, previously referred to. They may (Case X) or may not (Cases VI and VII) pulsate. Hirschboeck and Boman called attention to the radiolucence of the aneurysmal shadow in their case (Case IV, Fig. 3) and noted the visibility of lung markings through it. The roentgenograms of Cases III and X (Figs. 2 and 7) also showed this feature. One is probably not justified in ascribing differential diagnostic importance to the radiolucence of the shadow, because this appearance may be produced by other lesions. The size of the lesion and the type of exposure used probably have more to do with causing radiolucence than has the nature of the lesion.

In some cases (Cases I and VII, Figs. 1 and 4), part of shadow in the mediastinum may be due to an extravasation of blood into the mediastinal tissues, rather than to a sacculation of the aneurysm itself. Nonfatal rupture of the dissecting aneurysm into the mediastinum occurred in these 2 cases. Case XVII (Fig. 8) shows a similar phenomenon in a case of ruptured saccular aneurysm.

(*b*) In Case I (Fig. 1) a shadow is visible along the innominate artery, due to an extension of the dissection along that vessel. This appearance in the neighborhood of one of the large branches arising from the aortic arch is probably the most pathog-

FIG. 2. Case III. (Reported by Davy and Gates.[5] The authors have kindly permitted us to publish this roentgenogram.) A uniform, somewhat radiolucent widening of the shadow of the aortic arch and descending aorta is seen. This appearance, when considered in conjunction with the clinical features of the case, enabled the authors to make a diagnosis of dissecting aneurysm ante mortem.

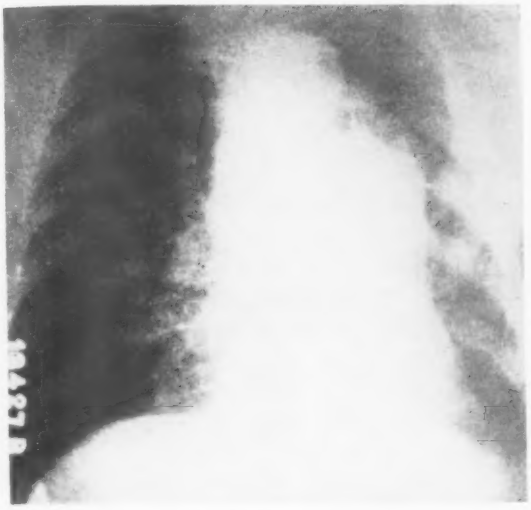


FIG. 3. Case IV. (Reported by Hirschboeck and Boman.[22] The authors have kindly sent us this photograph for publication.) A convex radiolucent shadow is visible extending to the left from the first part of the descending aorta. Roentgenoscopic examination revealed pulsation of this shadow. On the basis of the roentgenographic examination in this case, the authors made the diagnosis of dissecting aneurysm ante mortem. It was confirmed at necropsy.

nomonic roentgenological feature of dissecting aneurysm, but unfortunately it is relatively rare, for it did not occur in any of the other cases (except possibly Case VIII, Fig. 5).

(*c*) Patrick and Taylor reported that the aneurysmal mass in their case produced a partial obstruction of the esophagus and a marked displacement of this structure forward and to the left. This may not be a constant feature. It was not investigated in any other case. However, a consideration of the mediastinal anatomical relations suggests that it may be a relatively common occurrence. Displacement in either direction should be possible, but displacement to the right should be more frequent. A complete roentgenological examination of the heart and aorta should always include observation after the introduction of an opaque substance into the esophagus.

(*d*) The trachea was displaced to the right in 4 of our cases. In 2 (Cases I and IX, Figs. 1 and 6) this may have been due to fluid in the left chest pushing the entire mediastinum to the right. In the others (Cases VII and X, Figs. 4 and 7) it seems that the aneurysmal mass was probably responsible for the displacement.

(*e*) There has been a certain degree of cardiac enlargement in most of the cases, the cause of which has been discussed elsewhere.

(*f*) Fluid is very frequently present in the left pleural cavity (*vide supra*). Occasionally it is seen on the right (Case X, Fig. 7).

Roentgenography has certain limitations as a diagnostic procedure in dissecting aneurysm:

(*a*) If the presence of such a lesion is suspected, the patient should not be sent to the roentgen-ray department, since he

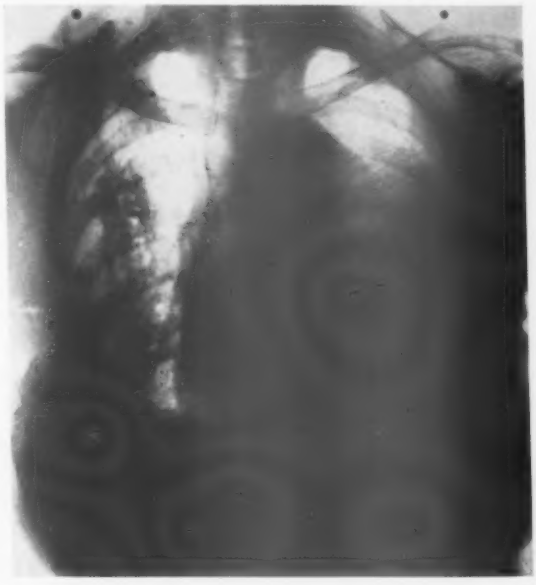


FIG. 4. Case VII. (Reported by Tidmarsh.[52] The author has been kind enough to send us this roentgenogram, and a large photograph of the pathological specimen.) The heart and trachea are displaced to the right. There is a diffuse shadow in the left lower lung field. A denser shadow is visible in the region of the aortic knob. These shadows are due to the dissecting aneurysm itself, to mediastinal and retropleural hemorrhagic extravasations, and to a left hydrothorax. The shadow in the right lung field is not related to the patient's present illness; it is due to an old calcified process in the right pleura.

FIG. 5. Case VIII. There is a diffuse widening of the shadow of the aortic arch. There is a shadow along the innominate artery, but we were unable to determine from the necropsy report whether or not it was due to an extension of the dissection along that vessel.

should be spared all unnecessary activity. Carefully supervised bedside roentgenography, however, would not seem to be contraindicated. At a later date, if the patient survives, it may be possible to transport him on a litter to the roentgen-ray department for further and more complete examination.

(*b*) The lesion may occasionally involve a part of the aorta which cannot be visualized by roentgenography.[32]

(*c*) It is usually impossible to differentiate accurately between the roentgenographic appearance of dissecting aneurysm and that produced by simple dilatation of the aorta, saccular aneurysm, and other mediastinal lesions. Moreover, occasionally dissecting aneurysm and saccular aneurysm occur in the same patient.[32] The findings are not pathognomonic in the average case, but they should be of very definite assistance in suggesting the correct diagnosis or in ruling out erroneous ones, if they are considered in conjunction with the clinical features of the case. It is probable that the use of the Potter-Bucky diaphragm in the roentgenographic study of these cases would aid materially in clear definition of the lesion.

It might be well to mention briefly a few of the commoner conditions with which dissecting aneurysm may be confused, without undertaking a complete discussion of the differential diagnosis.

(*A*) The most important of these is coronary thrombosis. Both lesions occur in the same group of individuals, from the standpoint of age and underlying circulatory pathological changes (hypertension and arteriosclerosis). The symptomatology of the attack (the type of pain, its duration, the accompanying shock, and the subsequent remission of discomfort) is apt to be strikingly similar. Subsequent fever and leucocytosis may occur in each.

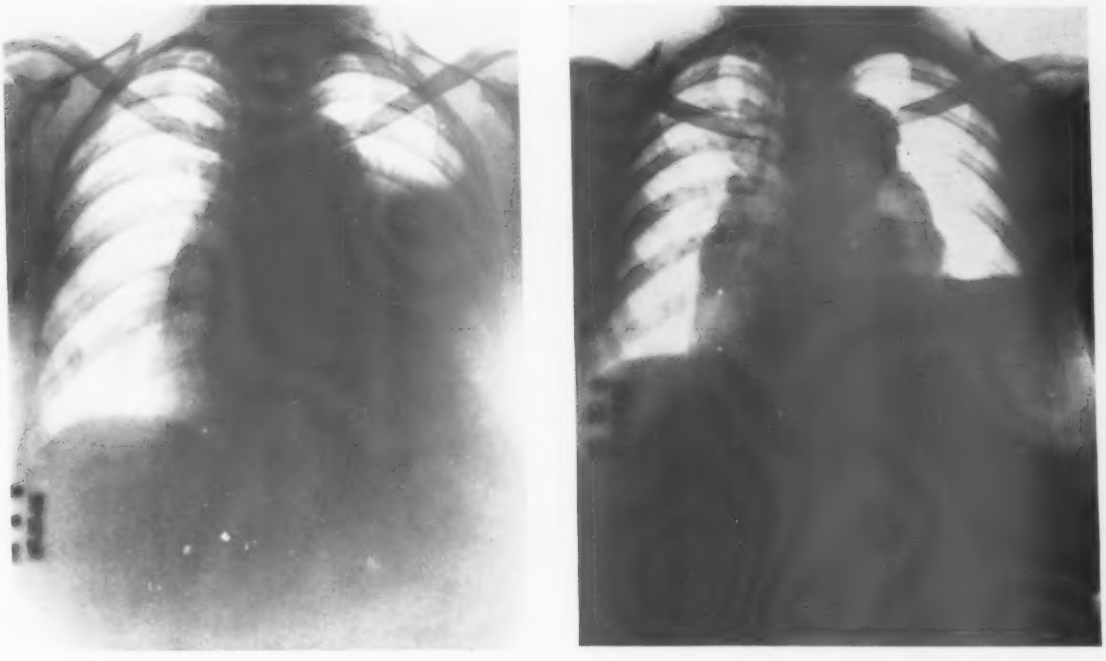
The important differential points might be enumerated as follows. (1) Dissecting aneurysm is more apt to occur during exertion or excitement. (2) The pain of dissecting aneurysm is more likely to show bilateral and widespread radiation; it occurs more frequently in the back; its occasional migratory character, mentioned above, should be an important differential point in some cases. (3) Anemia, unexplained aortic insufficiency and fluid in the left pleural cavity, following an attack of supposed coronary thrombosis, are findings suggestive of dissecting aneurysm. If this fluid is blood, one's suspicions should be definitely aroused. (4) A clue may sometimes be obtained from phenomena indicating pressure on the mediastinal structures (trachea, esophagus and veins). (5) Transient hemoglobinuria has been mentioned as a feature suggesting dissecting aneurysm.[20] (6) Electrocardiograms may be of diagnostic aid. Characteristic "coronary T-waves," or sequential changes in

the ventricular complex occurring over a relatively short period of time should favor the diagnosis of coronary thrombosis. However, one must not lose sight of the fact that these findings, supposedly pathognomonic of cardiac infarction, have been observed in other conditions,[15] including pericardial effusion. Moreover, a dissecting aneurysm might cause occlusion of a coronary artery by the same mechanism by which it causes occlusion of any other branch of the aorta. The "coronary T-waves" in Case VIII may be explainable on this basis. (7) of great importance in the diagnosis of dissecting aneurysm from coronary thrombosis are the phenomena which may appear as a result of the obstruction of the branches of the aorta by this lesion (*vide supra*). (8) The roentgenologic phenomena summarized in this paper should be of considerable aid in focusing diagnostic attention on the aorta rather than upon the coronary arteries.

(*B*) On account of the aortic diastolic murmur which develops in some cases, one might confuse dissecting aneurysm with rupture of an aortic valve.[23] In the latter condition, however, the murmur is apt to by much louder, sometimes audible at a distance from the patient.

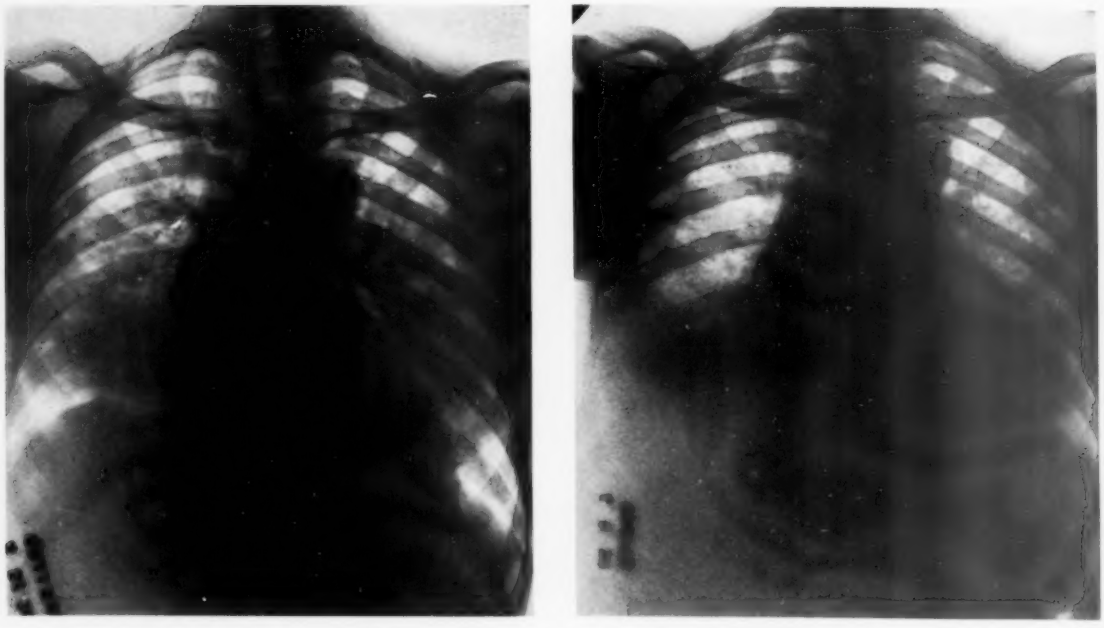
(*C*) A dissecting aneurysm may be mistaken for embolic obstruction of a large intrathoracic or intra-abdominal artery.

(*D*) The most difficult cases to diagnose are probably those in which there is no history of an acute painful onset to guide one's thoughts to the correct conclusion. These cases have usually been diagnosed myocardial insufficiency resulting from arteriosclerotic or hypertensive cardiovas-



A B

FIG. 6. Case IX. A. The left chest shows a dense shadow due to blood in the pleural cavity. The heart and trachea are displaced to the right. The shadow of the aortic arch is distinctly widened. B. After left-sided thoracentesis and the removal of 640 c.c. of blood, 700 c.c. of air was injected. There is a hydropneumothorax on the left. The mediastinal structures are displaced to the right. The shadow of the aortic knob is definitely widened, and just below, in the first part of the descending aorta, an arcuate excrescence is visible, extending from the aortic shadow into the shadow produced by the collapsed left lung. This excrescence coincided in position with a sacculation found in the aneurysm at necropsy.



A B

FIG. 7. Case X. A. Roentgenogram taken February 21, 1930. Cardiac enlargement and passive congestion of the lungs are evident. There is an arcuate shadow extending to the right from the ascending aorta, which on roentgenoscopic examination showed an expansile pulsation. At necropsy a dissecting aneurysm of the aorta was found with a huge sacculation in the region of this pulsating shadow along the ascending aorta. B. Cardiac enlargement, passive congestion of the lungs and pleural effusion at the rignt base are evident. The shadow along the ascending aorta is distinctly less arcuate than in A.

cular disease. In some cases, the cerebral phenomena have overshadowed the other aspects of the condition, and the correct but incomplete diagnosis of a cerebral vascular accident has been made (Cases XIII and XIV.)

(*E*) A dissecting aneurysm may be mistaken for certain intra-abdominal catastrophies,[22] such as a rupture of peptic ulcer or acute pancreatitis. The pain may also be mistaken for that of gallstone colic, or renal calculus.[20]

(*F*) It may give rise to the suspicion that a vascular accident has taken place in the spinal cord itself on account of the frequency with which symptoms referable to this structure occur.

If, however, one bears in mind dissecting aneurysm as a possible cause of sudden painful shocking circulatory accidents in patients past forty years of age, and is cognizant of its more common physical and roentgenographic manifestations one should be able to diagnose the lesion with much greater frequency than has been the case heretofore. Dissecting aneurysm is an admittedly rare lesion but when tempted to dismiss it as a pathological curiosity, entirely devoid of other than academic interest, one should remember that there have been 8 cases (and possibly more) in the Philadelphia General Hospital during the past three years.

SUMMARY

The subject of dissecting aneurysm of the aorta has been reviewed from the standpoint of its historical aspect, etiology, pathological anatomy, clinical features and diagnosis.

Six previously recorded cases are briefly reviewed, and 11 new cases with necropsy (4 with roentgenograms of the chest) are reported.

A diseased vessel wall is an essential prerequisite to the formation of a dissecting

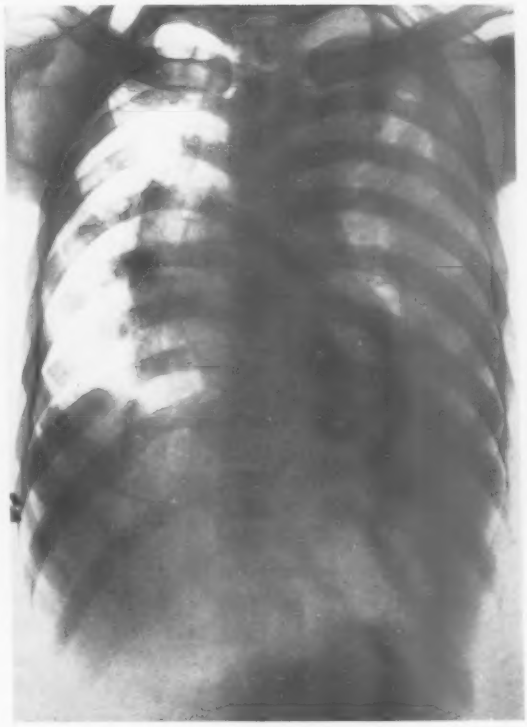


Fig. 8. Case xvii. The saccular aneurysm, located below the diaphragm has eroded the lower thoracic and first lumbar vertebrae and the eleventh rib. The heart and trachea are displaced to the right. There is a diffuse shadow in the left chest. A denser shadow is visible, extending upward and to the left from the region of the aortic knob. There is a shadow to the right of the heart. All these are probably due to hemorrhagic extravasations.

aneurysm. Hypertension and arterio-sclerosis are probably its most important predisposing antecedents. Exertion is often the exciting cause. Syphilis is probably not an etiological factor. Neither does syphilitic aortitis seem to prevent the formation of a dissecting aneurysm.

Fifty per cent of all cases die from rupture of the aneurysm into the pericardium.

Probably about 20 per cent of cases undergo internal rupture (perforation of the aneurysm back into the aortic lumen) and in about half of these cases "healing" occurs, i.e., death is delayed for an indefinite period, and when it does take place, it is due to some cause other than rupture of the dissecting aneurysm. Occasionally patients survive for twenty or thirty years.

The formation of a dissecting aneurysm is frequently, but not invariably, accompanied by agonizing pain and shock.

In the majority of cases, death does not take place immediately, there being an intervening period during which the symptoms may entirely subside. Diagnosis of the lesion is possible at this time.

The important diagnostic signs are: fever, leucocytosis, anemia, an aortic diastolic murmur, certain phenomena due to obstruction of one or more of the aortic branches by the mass of the aneurysm, signs of spinal cord dysfunction, signs of pressure by the aneurysm on the mediastinal veins or retroperitoneal sympathetic ganglia, fluid (often bloody) in the left pleural cavity, and a palpable mass along the course of the aorta.

Transient hemoglobinuria may be a sign of importance but needs further investigation before it can be accepted as such.

The *roentgenographic features* of dissecting aneurysm are illustrated by figures from 3 previously reported cases and from 4 new ones:

(*a*) There is a deformity of the supracardiac shadow. Frequently an arcuate excrescence extends outward from some point in the aortic arch. This shadow may or may not pulsate.

(*b*) The shadow produced by an extension of the dissection along one or more of the large branches of the aortic arch is probably the most pathognomonic roentgenological sign.

(*c*) Displacement of the esophagus and trachea may occur.

(*d*) The heart is usually enlarged.

(*e*) Fluid is frequently present in the left pleural cavity. Non-fatal rupture of the aneurysm into the left pleural cavity occurs quite often in these cases.

(*f*) Non-fatal rupture of the aneurysm into the periaortic tissues may produce a bizarre roentgenographic appearance.

Roentgenography is limited in certain respects in its diagnostic usefulness, especially on account of the fact that the

roentgenographic findings in cases of dissecting aneurysm are not usually pathognomonic. They may, however, be of very definite diagnostic importance when considered in conjunction with the history and physical findings. This is especially true because the lesions with which a dissecting aneurysm may be confused from the roentgenographic standpoint (aortic dilatation, aneurysm, mediastinal abscess and tumor) comprise a different group from those with which it is most likely to be confused on clinical grounds (coronary occlusion, embolus, spinal cord lesions, and intra-abdominal catastrophies).

Dissecting aneurysm constitutes one of the rarer, sudden, painful, shocking, circulatory complications of arteriosclerotic and hypertensive cardiovascular disease in patients past forty years of age, which is apt to be temporarily survived.

The fact that it was found at the necropsy table 8 times in the past three years in one Philadelphia hospital would seem to indicate that it is not a mere pathological curiosity entirely devoid of practical importance.

CASE REPORTS

Case 1. J. R., a colored male, aged sixty-three, was admitted to the Hospital of the University of Pennsylvania, January 23, 1927, on the service of Dr. Alfred Stengel. He had felt perfectly well until January, 1926. At that time he began to notice weakness and dyspnea on exertion, which became progressively more severe. He also experienced palpitation and mild substernal pain as a result of activity. He had nocturia, dysuria, frequency and urgency, and suffered from occasional frontal headaches. He had had attacks of epistaxis. During the three months prior to admission he noticed a sense of fullness in his throat. He was occasionally troubled with eructations and constipation. There was no history of any acute attack of pain, dyspnea, syncope or any record of injury. An attack of typhoid fever "long ago" was the only significant feature in his past medical history. He denied having had any venereal disease. The patient's father and mother had died from causes unknown to him. His wife had had no miscarriages and was living and well at fifty-four. There were 9 sons and 7 daughters; 3 other children had died in infancy. He described his consumption of alcohol and tobacco as moderate. He had worked as a blacksmith all his life, but had had to give up his occupation in December, 1926, on account of the symptoms referred to above. During January, 1927, the slightest exertion caused extreme dyspnea.

Physical examination on admission was recorded as follows: The patient was a poorly nourished, markedly dyspneic colored male of sixty-three years. The temperature was 103°F. The pupils were equal and reacted to light; the eyegrounds showed considerable vascular sclerosis. Dental and tonsillar infection were evident. There were no masses noted in the neck. No tracheal tug was elicited. There was slight prominence of the right clavicle and first rib. Venous engorgement was manifest over the left upper chest and in the left arm. The percussion note over the left lower chest was flat as high as the fourth rib; tactile fremitus was diminished over this non-resonant area, but tubular breathing was heard. The right side of the chest was clear. The blood pressure in the left arm was 230 systolic and 105 diastolic, in the right arm 210 systolic and 105 diastolic. The pulse showed an extrasystolic arrhythmia and ranged between 70 and 95 beats per minute. There was moderate cardiac enlargement, and a widened supracardiac dullness. At the apex a blowing systolic murmur was heard, and at the aortic area both a systolic and a diastolic murmur were audible. The aortic second sound was accentuated and ringing. The abdomen was quite distended; the liver was described as barely palpable and not tender. There was no edema; peripheral sclerosis was well marked; all tendon reflexes were decreased in amplitude.

Laboratory studies were recorded as follows: Urine: amber with flocculent sediment, specific gravity 1.009 (remaining fixed at about that level), acid reaction, a heavy trace of albumin (later a cloud was reported), no sugar; microscopic examination of the sediment showed many heavy granular casts, no erythrocytes, and many leucocytes. Blood count: erythrocytes 2,960,000, leucocytes 19,400, hemoglobin (Sahli) 40 per cent; differential: neutrophiles, 91 per cent; lymphocytes, 7 per cent; large mononuclears, 2 per cent. The Wassermann reaction of the blood was negative, but the Kahn reaction was medium positive. Phenolsulphonphthalein elimination after intramuscular injec-

tion was 4 per cent in two hours. Blood urea nitrogen was 114 mg. per cent. The electrocardiogram showed occasional ventricular extrasystoles, a tendency toward left axis deviation, and inverted T-waves in Lead I. A roentgenogram of the chest was made on January 25, 1927 (Fig. 1), and was reported as showing a left pleural effusion, displacement of the heart to the right, *and an appearance suggesting aneurysm of the innominate artery.*

During the patient's three days' stay in the hospital the temperature dropped from its original level of 103°F. and remained between 98° and 99.8°F. In other respects there was little change noted in his condition. Various diagnoses were made: aneurysm of the aorta, mediastinal neoplasm, severe hypertensive cardiorenal disease, etc. Death occurred rather suddenly and unexpectedly at 10 A.M., January 26, 1927.

Necropsy was performed by Dr. B. Lucke on the day of death. A dissecting aneurysm involving the entire length of the aorta was found. The initial rupture was a small ragged tear in the intima at the junction of the descending arch and the thoracic aorta. From this point the blood had entered the media, and had dissected back to the semilunar valve as well as down to the aortic bifurcation. The dissection had extended out along the innominate artery. A mass of organized blood was found in the substance of the media of that vessel which had produced the shadow along the innominate artery in the roentgenogram (see Fig. 1). The blood in the aortic walls averaged 2 cm. in thickness. It was clotted and firmly organized around the ascending and transverse arch. Elsewhere the clots were more recent. There was no mention, in the necropsy report, of a point of reëntrance of the aneurysm into the aortic lumen.

The adventitia was torn through in several places, the largest tear being on the posterior surface of the aorta in the midthoracic region. Several of these tears were from 2 to 3 cm. in length. The blood which escaped through them lay between the external aspect of the adventitia and the great serous membranes in the thorax and abdomen. Some had also infiltrated the mediastinal tissues in the neighborhood of the ascending and transverse portions of the aortic arch. The largest amount of blood lay behind the aorta in the lower part of the thorax and in the upper part of the abdomen. A clot the size of a grapefruit was situated behind the pancreas.

The intima of the aorta showed numerous hyaline and atheromatous plaques, and just above the initial tear of the intima there was a small atheromatous ulcer; another was situated in the posterior wall of the abdominal aorta about 5 cm. above its bifurcation. In the arch there were a number of pitted areas which contrasted with the neighboring hyaline and atheromatous plaques. The heart showed hypertrophy (weight, 500 gm.) and coronary arteriosclerosis. There was no valvular lesion, despite the aortic regurgitant murmur noted on physical examination (cf. Resnik and Keefer[45] and Lundberg[30]). A left-sided, blood-stained hydrothorax was present. The kidneys showed advanced arteriolar nephrosclerosis. The spleen showed a moderate amount of fibrosis.

Histological examination showed the dissecting blood lying between the outer layers of the media.[43] It was organized in many places, but there was no intima lining the aneurysm. There were areas of typical syphilitic aortitis as well as signs of advanced arteriosclerosis.

Dr. Lucke summarized his impression of the situation as follows in his pathological report: "Rupture of either an atheromatous ulcer or of a small gumma occurred in the descending arch. Through this tear blood forced its way and dissected the aortic walls. At a later date, a small rupture of the adventitia occurred, and a relatively small quantity of blood infiltrated the mediastinal tissues. The final rupture occurred in the adventitia of the midthoracic portion, with the escape of a massive amount of blood into the tissues behind the peritoneum and pleura."

CASE II. (Reported by Goodman.[17]) D. F. D., a white male, aged forty, was admitted to the Medical Wards of the Presbyterian Hospital, Philadelphia, on July 15, 1913. On July 14, while carrying two valises and running for a train, he was seized with severe dyspnea, vomiting and diarrhea. On admission he was pallid, and complained of pain and oppression in the left side of the chest. The Wassermann test of the blood was negative. On the day of admission 650 c.c. of blood was removed from the left pleural cavity by thoracentesis. After this his discomfort disappeared. On July 18 a roentgenogram of the chest was made, and showed a "dilated aorta and hemothorax." After a brief febrile illness the patient left the hospital on July 27. Soon after discharge he was examined roentgenoscopically by Dr. Graves of the Hospital of the University of Pennsylvania, at which

time "a large diffuse aneurysm of the aorta could be plainly seen. The left chest showed a dense shadow due to hemothorax." On November 28 the patient was roentgenoscoped once more, and showed an "aneurysm of the arch of the aorta, previously ruptured, with left-sided hemothorax, now cleared up, leaving pleural thickening." He was then lost sight of.

Unfortunately all roentgenograms of this case have been destroyed, and since there was no necropsy to verify Goodman's diagnosis of dissecting aneurysm a certain element of doubt exists as to the exact nature of the lesion. Nevertheless, after corresponding with the author and reviewing the original records at the Presbyterian Hospital, we believe that Goodman's diagnosis was justified, and that this was the first case on record in which a dissecting aneurysm was studied roentgenographically.

CASE III. (Reported by Davy and Gates[5]). A man, aged fifty-five, with a long-standing hypertension noticed a discomfort in his chest one morning (November 1, 1921), stretched his arms backward, and was seized with intense pain in the region of the heart. He was restless and shocked. The pain radiated through from the left nipple in front to a point at the same level near the spine behind. Physical examination showed dullness to percussion in the left lower chest posteriorly. The next day a diagnosis of probable dissecting aneurysm was made on clinical grounds, and confirmed by a roentgenogram made by Dr. Hodgson (Fig. 2). (Dr. Gates has been kind enough to send us the original film for publication in this review.) The patient lived for nineteen days after the original accident. On November 19, 1921, while apparently comfortable, he was seized with a sudden attack of faintness and died almost immediately. Necropsy showed both old and fresh blood in the left pleural cavity. "There was a definite dissecting aneurysm about the size of a small orange at the apex of the transverse arch of the aorta, caused by a rupture of the inner coats of the aorta at this situation, where it was definitely weakened by calcareous patches of atheroma. The dissecting aneurysm had ruptured into the left pleural cavity, and so caused the death from hemorrhage."

This is the first clear-cut case in which a roentgenogram was made and the diagnosis of dissecting aneurysm confirmed by necropsy.

CASE IV. (Reported by Hirschboeck and Boman[22]). A man, aged sixty-three, was seized, on November 21, 1921, with sudden severe pain in the upper abdomen followed by vomiting and shock. The fever (101°F.), leucocytosis (14,000), and board-like rigidity of the abdominal muscles led his physician to consider the advisability of an exploratory laparotomy. A definite improvement, however, was manifest the next day, and the patient left the hospital five days later, having apparently recovered from his attack. On December 24 he was seen once more, and at this time complained of a good deal of dyspnea and moderate upper abdominal pain. There was dullness to percussion at the base of the left lung. A roentgen examination (Fig. 3) was made at this time. (Dr. Hirschboeck has been so kind as to send us a photograph of the original plate for publication.) The roentgen report reads as follows:—"We found an extremely large shadow to the left of the heart .... The shadow was radiolucent and not of uniform density, extending from a point corresponding to the top of the arch of the aorta to the left, outward and downward, nearly to the periphery, forming an obtuse angle and then turning directly downward toward the diaphragm, where its further course could not be traced on account of its fusion with the denser subdiaphragmatic structures. The unusual radiolucence, with the aortic shadow obviously extending down along the inner portion of this shadow, and the visualization of the hilus markings through it, were considered unusual, and various diagnoses were considered." It was concluded that "no other condition except a dissecting aneurysm which had undergone recanalization, at times free of blood, would permit of this curious radiolucent appearance."

The patient subsequently grew worse, developed signs of fluid in the left pleural cavity, circulatory embarrassment, and mental disturbances. He gradually weakened, and died suddenly on March 19, 1922.

At necropsy it was found that death had been due to hemorrhage into the left pleural cavity. This space was full of clotted blood. A dissecting aneurysm was found, beginning at the arch of the aorta and extending its entire length to the femoral vessels on either side. There was an out-pouching of the aneurysm just below its origin, which probably accounts for the shadow which is most conspicuous in the roentgenogram (see Fig. 3). Through this sacculation it had ruptured into the left pleural cavity. The inner surface of the aneurysm was described as smooth and shiny. Evidence of luetic aortitis were present, despite a negative blood Wassermann reaction.

CASE V. (Reported by Hall.[19]) In 1910, a white male, aged seventeen, suffered from severe colicky pain in the epigastrium two days after running a mile race. His physician diagnosed "serious valvular disease of the heart," and confined him to bed for six months. At the end of that time he was able to engage in strenuous physical exercise (swimming, riding, etc.) without distress, and remained free of

cardiac symptoms for fifteen years. In February, 1925, he developed signs of congestive heart failure which advanced rather rapidly. He was found to have aortic insufficiency and a rough systolic aortic murmur. A roentgenogram of the chest showed "a marked enlargement of the heart of the aortic type, enlargement of the root of the aorta, and passive congestion of the lungs." Hospital treatment improved the condition temporarily, but he soon relapsed and died of congestive heart failure in August, 1925.

Necropsy revealed evidences of marked passive congestion of the various organs. There was no free fluid in the pleural cavities. The heart was markedly enlarged. The aorta showed a dissecting aneurysm the size of a man's fist, which began 1 cm. above the valve and reëntered the aortic lumen at the attachment of a patent ductus arteriosus. It appeared that practically all the blood from the left ventricle had to pass through the aneurysmal cavity. The sac was lined with endothelium. The aortic valve leaflets appeared normal despite the aortic diastolic murmur which had been heard on physical examination.[30]

Case VI. (Reported by Patrick and Taylor.[41]) A patient, aged thirty-two, entered St. Thomas Hospital with the chief complaint of dysphagia. There was no history of acute or painful onset, and no demonstrable arterial disease. "An x-ray of the chest showed a shadow passing out from the mediastinal shadow into the left lung area at the level of the bodies of the 4th to 8th dorsal vertebrae. No pulsation could be seen in the shadow. On swallowing opaque fluid, it was seen that the oesophagus was displaced forwards and to the left, being stretched over the tumor, and thereby partly obstructed." (Unfortunately the roentgenogram has been destroyed.) The patient had a leucocytosis of 17,000, and the roentgen report, probably colored by this finding, stated "large mediastinal tumor displacing the oesophagus forwards and to the left—appearance of mediastinal abscess." An operation was performed; severe hemorrhage was encountered; an aneurysm was suspected. Several days later the patient died. At necropsy a large dissecting aneurysm was found, extending from the arch to the bifurcation of the aorta, giving the appearance of a double aorta. Dr. Patrick says in his letter, "The X-Ray did not help us to the diagnosis of aneurysm in this case, as no pulsation was visible on the screen; the question of aneurysm was hardly entertained until the first operation revealed it, and the fact that the aneurysm was of the dissecting type was not discovered until the autopsy."

Case VII. (Reported by Tidmarsh.[32]) A woman, aged sixty-nine, during mild exertion was seized with sudden severe pain between the shoulders, felt chilly and vomited. The next day percussion dullness, distant breath sounds and râles were heard at the left base. The acute pain had subsided and had been replaced by a dull ache in the left chest posteriorly. On the sixth day the patient suffered a sudden, severe boring pain through the left chest, aggravated by respiration. She was admitted to the hospital at that time. The temperature was 98°F.; the pulse rate, 90; and the respiratory rate, 24. The blood pressure was 130 systolic and 60 diastolic. The patient was slightly cyanosed, but not in apparent distress. There was a rough apical systolic murmur, otherwise the heart sounds were normal. Physical examination of the chest showed a lagging of respiratory movement on the left. There were diminished tactile fremitus, a flat percussion note, suppressed breath sounds and râles at the left base. In other respects the examination proved negative. The only finding of note in the laboratory studies was a blood count showing 3,860,000 erythrocytes, 14,700 leucocytes and 70 per cent hemoglobin. A blood Wassermann test was not done; the patient denied having had any venereal disease. A roentgen examination (Fig. 4) of the chest was reported as follows: "Heart and trachea are displaced to the right. Aorta obscured by a shadow in that region the size and shape of a kidney, with its upper limit at the left sternoclavicular junction. No pulsation was seen on fluoroscopic examination. Left diaphragm is almost obscured by a homogeneous density extending upwards and fading into normal radiability at the level of the 5th space behind. Appearance here is that of a layer of fluid in the pleural cavity. Thickened pleura, left apex. Right diaphragm is not obscured. There is a large irregular calcified pleural plaque in the right chest, extending from the diaphragm upward to the fifth space, and involving the middle and inner zones of the pleura. Laterally it is sharply limited by a vertical straight line, showing normal lung markings from there to the lateral wall of the chest." (This finding in the right chest apparently had nothing to do with the present illness.)

A thoracentesis was done, and 2 c.c. of dark red blood aspirated. On the tenth day, a complete right-sided hemiplegia developed. Death occurred on the twelfth day of illness.

Necropsy showed a dissecting aneurysm of the arch and descending part of the aorta. The large branches were not involved in the dissection. The intimal perforation as well as the site of external rupture was in the first part of the descending aorta. The blood which had escaped through the rent in the adventitia had infiltrated the neighboring tissues and had collected behind the parietal pleura in the left chest wall posteriorly. There was a left hydrothorax which had produced a collapse of the left lower lobe. The calcified lesion in the anterior right pleura was an old process. There was a clot in the left internal carotid artery, and softening in the left side of the brain. The aorta showed syphilitic infiltration. Generalized arteriosclerosis was present. The kidneys presented an unusual picture of multiple recent infarction with intense hemorrhagic extravasations.

In a personal communication, the author says:

"While I have no definite proof of the following statement, it is my belief that the aneurysm formed at the time of the original pain and that the external rupture occurred with the second attack of severe boring pain on the sixth day. This opinion was also expressed by the pathologist and other physicians, when we discussed this case at pathological conference."

Case VIII. H. H., a white female, aged sixty-two, was admitted to the Philadelphia General Hospital on September 17, 1928, on the service of Dr. Allyn. She had lost weight rapidly during the past Summer, and had suffered with "neuritis," for which she had been taking osteopathic treatments. For two weeks prior to admission she had experienced extreme weakness, palpitation, and moderate pain behind the orbits. She was constipated, and had vomited once recently. There had been no edema nor precordial pain. There is no mention of a severe attack of any sort which might be considered the onset of the dissecting aneurysm. Her past history contains mention of scarlet fever, diphtheria, and mumps in childhood. At the age of forty she had had pleurisy. There was no history of venereal infection. During her adult life she had experienced good health and had worked hard. Her family history states that several of her relatives had had "strokes."

Examination on admission showed a normal temperature, and a respiratory rate of 24. The patient was described as a small, thin, gray-haired women. The conjunctivae were inflamed, the pupils normal. The cervical veins were engorged and showed exaggerated pulsation; there was no thyroid enlargement. The chest was thin and flat; the right base posteriorly was slightly impaired to percussion, but there were no râles. The blood pressure was 110 systolic and 60 diastolic in both arms. The radial pulse was of small volume but appeared stronger to touch on the right. It was irregular, with a rate of 84. The heart rate at the apex was 144. The aortic dullness was increased to left and right, and a pulsating mass was palpable in the episternal notch. The heart was described as slightly enlarged. A blowing apical systolic murmur was heard, and the aortic second sound was of greater intensity than the pulmonic second. All peripheral vessels were moderately sclerosed. There were no significant findings recorded in the rest of the physical examination.

During the patient's stay in the hospital the heart rate came down to 84 and the pulse deficit disappeared. No further progress notes were made until the final one (Sept. 29, 1928), "patient growing weaker, pulse fairly regular, and occasional extrasystole. However, patient seems very tired." The manner of her death (Sept. 30, 1928) is not recorded.

Laboratory findings were as follows: Urine analysis (Sept. 18, 1928): clear, acid, specific gravity 1.015, no albumin, no sugar, a few leucocytes. Blood count September 28, 1928: erythrocytes 3,530,000, leucocytes 12,900, hemoglobin 76 per cent; differential: neutrophiles 81 per cent, lymphocytes 19 per cent. The Wassermann test of the blood (Sept. 20) showed a strongly positive reaction. Blood sugar was 87 mg. per cent. Blood urea nitrogen was 27 mg. per cent. The eye-grounds examination showed nothing significant, but a ciliary flush was noted by the ophthalmologist. Two electrocardiographic tracings taken on September 18 and 21 were much the same in appearance, showing occasional ventricular extrasystoles, flat T-waves in Lead I and an elevation of the S-T interval in Leads II and III. The irregularity which was present on admission might have been due either to frequent extrasystoles, or to a paroxysm of auricular fibrillation. A roentgenogram of the chest (Fig. 5) was made on September 19, 1928, eleven days before death, and was reported as follows: "There is a dilatation of the ascending, longitudinal and descending branches of the aorta, slight atheroma of the arch, passive congestion of the lungs, aortitis. Aneurysm is questionable, although a small one may be present."

Necropsy performed on September 30 was reported as follows: "The first portion of the aorta shows a dissecting aneurysm which has ruptured into the pericardial sac. The rest of the aorta is markedly atheromatous and contains numerous ulcers throughout, some of which also show a tendency to the formation of dissecting aneurysmal process. The heart weighs 810 grams, and shows marked hypertrophy. The pericardium contains 120 c.c. of blood. The surface of the heart and inner surface of the pericardium show a fibrinous exudate which is shaggy and blood-stained. The aortic valve is competent, and the leaflets atheromatous."

It is barely possible that the dissecting aneurysm in this patient developed at some time after the roentgenogram of the chest was made; possibly it formed just

before she died. However, the appearance of the aorta in the roentgenogram, the presence on admission of a pulsating mass in the suprasternal notch, the fibrinous exudate over the surface of the heart, the leucocytosis and anemia and the absence of any record of a sudden change in the patient's condition from the time of admission until death, mitigate against this possibility.

Case IX. M. P., a colored female, aged fifty-five, was admitted to the Philadelphia General Hospital, May 8, 1930, on the service of Dr. Loewenberg. For the past few years she had been dyspneic on exertion, but had been otherwise apparently healthy. On Sunday, May 4, 1930, while riding on an elevated street car, she was seized with sharp shooting pain of great intensity, which began in the back between the shoulder blades and radiated forward on the left side beneath her heart, into the left upper abdomen and then down the right leg. She felt very ill, and vomited. On attempting to rise she found that her right leg would not support her. Her husband dragged her bodily from the car, and she "crawled like a baby down the L. steps to a taxi." On arriving home she went to bed and stayed there until the day of admission. The pain subsided at times, but kept recurring in paroxysms accompanied by nausea, vomiting and restlessness. At first the right leg from the knee to the toes had been the seat of "scrutinizing pain"; later it became cold and numb. Since the onset of the present illness she had experienced difficulty in starting her urinary stream, and some discomfort had attended micturition. No history of hematuria was found in her record.

The past medical history contained mention of rheumatic fever, tonsillitis, diphtheria, typhoid fever and malaria during childhood. She had had pneumonia at the age of thirty-two, and influenza at forty-four. She had been delivered of a still-born child, and had had no subsequent pregnancies. Her menopause occurred in 1923. She had never used alcohol or tobacco.

On admission, she was described as an obese black woman who was very restless and perspired freely. She was not definitely dyspneic. The pupils were slightly irregular, equal, and reacted normally. She had false teeth and cryptic tonsils. The neck showed forceful pulsations, and the head nodded with each heart beat. The thyroid gland was not palpable; the trachea was displaced slightly to the right. There were signs of a large amount of fluid in the left pleural cavity. The apex beat of the heart was in the fifth left interspace 11 cm. from the midsternal line. The right border was percussed 3 cm. from the midsternal line. Auscultation revealed a soft systolic murmur, heard best near the sternum in the fourth interspace but audible at the apex. The aortic second sound was accentuated. The peripheral blood vessels were thickened. The blood pressure was 180 systolic and 95 diastolic, the pulse 110, the respirations 30, and the temperature 99°F. There was some tenderness in the epigastrium; resistance, probably due to hepatic enlargement, was encountered in the right upper quadrant. The extremities were not edematous. There was no pulsation in the right femoral artery, nor in any of the arteries of the right lower extremity. The strength in this member was impaired, and the tendon reflexes were absent, but there was no Babinski's sign nor ankle clonus. Perception of pain and touch was markedly diminished up to the middle of the calf. Sense of position was lost in the foot, but temperature perception was normal.

During the patient's stay in the hospital the temperature varied from 99° to 101°F., the pulse rate remained at approximately 110 beats per minute, and the respirations varied from 25 to 40 per minute. The blood pressure varied from 130 systolic and 80 diastolic in both arms to 180 systolic and 110 diastolic in the right arm, and 175 systolic and 100 diastolic in the left. The pulse in the right femoral artery never returned. The patient complained of pain in the right leg from time to time. On May 9, 1930, a thoracentesis was done, and 100 c.c. of blood was aspirated from the left pleural cavity. More fluid was present but it could not be removed because the needle became obstructed by clots. On May 16 the thoracentesis was repeated; 640 c.c. of blood was aspirated, and 700 c.c. of air injected. The resident physician who performed the operation described feeling something solid with the point of the needle. On May 19, the patient was quite weak, but in other respects her condition was similar to that presented on admission. That evening, while eating her supper, she died suddenly.

Laboratory reports: Urine analysis: amber color, acid reaction, specific gravity 1.020, a trace of albumin, no sugar; a few leucocytes,

hyaline casts and epithelial cells. (Several other urinalyses were much the same, except that on May 11 a few erythrocytes were reported.) Blood count (May 9): erythrocytes 3,260,000; leucocytes, 12,800; hemoglobin, 50 per cent; differential count: neutrophiles, 78 per cent; lymphocytes, 12 per cent; large mononuclears, 10 per cent. Blood sugar (May 10) 110 mg. per cent. Blood urea nitrogen 23 mg. per cent. Blood Wassermann reaction negative with both antigens. Two roentgenograms of the chest were taken. The first (May 9) (Fig. 6 A) was reported as follows: "There is a left-sided pleural effusion. Heart and trachea displaced to right. Effusion extends to the fifth rib anteriorly." The second (May 10) (Fig. 6 B), taken after thoracentesis and the injection of 700 c.c. of air, was reported as follows: "Hydropneumothorax, left side, displacing heart and trachea to right and compressing left lung. There is no evidence of aneurysm. We are unable to demonstrate the presence of a new growth."

A necropsy was performed by Dr. E. B. Krumbhaar on May 20, 1930. "On opening the thorax a large amount of bloody fluid and coagulated blood were found in the left pleural cavity. The bloody fluid has a brownish color as if it had been there for some time. The large blood clot is of currant jelly type, and is a fairly accurate mould of the pleural cavity, weighing 1,640 grams. The heart weighs 440 grams, and is large and flabby. The valves are all essentially normal. The muscle is of normal color and consistency. The coronary vessels are slightly sclerotic but have normally wide lumina. The aorta: Opposite the lines of closure of the aortic valves there is a mild yellowish thickening for 10 to 15 cm. The ascending portion is practically normal; at this point there begin to appear raised gray and yellow areas, interspersed with a moderate amount of puckering, and it is seen that the middle coat is easily separated from the outer coat and that a small amount of blood occupies this space. As the arch and descending portions are examined, separation between the coats becomes more marked, and a considerable amount of currant jelly clot lies between the layers of the aortic wall. On the external surface of the thoracic portion of the aorta there is a bulging granular area 1×6 mm. There is a matted mass of fibrin several millimeters in thickness over this area, and no gross opening can be found, but this is probably the site of the external rupture of the dissecting aneurysm through which blood escaped into the pleural cavity. The dissecting blood does not extend into any of the vessels of the neck. To the left of the left subclavian artery is a semilunar opening 1 cm. long which is occupied by a currant jelly clot, partly in the true lumen of the aorta, but connecting with the mass in the dissected cavity. It is obvious that this is the beginning of the dissecting aneurysm and that some blood happened to clot in the opening at the time of death. The dissecting aneurysm is easily followed down the entire length of the aorta and iliac vessels, where it is lost beneath the groin. Examination of the femoral arteries shows them to be normal and without dissecting aneurysm.

"In the thoracic and abdominal portions, the aorta only shows a moderate amount of puckering, and raised gray areas which are but slightly suggestive of syphilis. There is considerable sclerosis of the Monckeberg's type in the celiac axis and mesenteric arteries. Over the iliac artery and extending into the pelvis there are retroperitoneal hemorrhagic extravasations proceeding from the dissecting aneurysm. The liver shows a moderate amount of cloudy swelling. The kidneys show benign arteriolar nephrosclerosis. There is a right-sided ovarian cyst."

This dissecting aneurysm extended from the junction of the ascending and transverse portions of the arch of the aorta to the iliac vessels, disappearing in the region of Poupart's ligament on each side. It encircled about half the circumference of the aorta throughout its length. There was a sacculation posterolaterally at the junction of the transverse and descending portions of the aortic arch. The lumen of the right common iliac artery was obliterated by a mass of clotted blood in the dissecting aneurysm. The left common iliac artery was partially obstructed in a similar fashion. The original intimal rupture was near the orifice of the left subclavian artery. External rupture into the left pleura probably occurred in the upper part of the thoracic aorta. The origin of the hemorrhagic extravasations in the pelvis is not stated, and could not be determined from the preserved specimen.

Case x. N. W., a colored male, aged twenty-eight, entered the Philadelphia General Hospital, February 20, 1930, on the service of Dr. Loewenberg. He had been well six weeks previously and had taken a trip to South Carolina. After having been there about a week he became very intoxicated and "caught a cold"

which became progressively worse. During the week prior to admission he suffered from severe night sweats, headache, anorexia, oliguria, epistaxis, persistent productive cough, hematemesis, indigestion and weakness, and he said that he had lost a considerable amount of weight. There is no mention of an attack of severe pain, dyspnea or unconsciousness which might have signalled the onset of a dissecting aneurysm, but his perception of sensory stimuli and his memory were probably somewhat obtunded during his visit to South Carolina, so this negative history should be accepted with a certain amount of reservation.

The past medical history contains mention of a chancre in 1923 and "rheumatism," following which "his heart became bad." He had had a Neisserian infection in 1928.

He had worked as a stevedore for nine years. His consumption of alcohol and tobacco was quite excessive.

He had been married in 1924 and had one child aged six. His wife had had one miscarriage. His mother died at the age of forty of "nephritis." The family history is otherwise uncertain or irrelevant.

Physical examination: The patient was perspiring freely on admission. There was a persistent cough, productive of blood-tinged mucopurulent sputum. He was moderately well nourished. There was slight exophthalmos, but in other respects the ocular examination was negative. The face was symmetrical, the pharynx was red and congested, and the tongue showed a fine tremor. There were enlarged lymph nodes in the posterior cervical chain. Marked pulsations were visible in the cervical vessels and in the suprasternal notch. The chest examination showed decreased aeration, impairment, and râles throughout the right lung area. The left side was hyperresonant, but otherwise normal. The apex beat of the heart was heaving in character, and located in the sixth interspace, anterior axillary line. There was a blowing apical systolic murmur, which could be heard in the left chest posteriorly. At the aortic area a metallic systolic squeak and a diastolic murmur were heard. The blood pressure was 150 systolic and 90 diastolic in the left arm, and 145 systolic and 85 diastolic in the right arm. The pulse was collapsing in type. Examination of the abdomen showed possible enlargement of the liver, no tenderness, and no masses. There was inguinal and epitrochlear adenopathy. The extremities were otherwise negative.

The patient gradually improved. The pulmonary signs cleared up, and he was discharged on March 25, 1930. There had been a moderate amount of fever—up to 101°F. at first, but this had gradually subsided. The pulse had varied from 80 to 90, and the respirations from 20 to 30.

He was re-admitted on April 4, 1930, complaining of insomnia, cough, and moderate dyspnea. There were no signs of decompensation. He was not considered ill enough to stay long, so he was discharged.

He came in once more on June 5, 1930. At this time he showed signs of heart failure, with dyspnea, edema, basal râles, engorged cervical veins and hepatic enlargement. The increased width of aortic dullness, the transmission of the apical systolic murmur to the back, the cervical venous distention and the blood pressure findings made the attending physicians suspect aneurysm of the aorta. The blood pressures were recorded as follows: Right arm 130/40, left arm 140/40, right leg 280/80, left leg 220/70. During the patient's stay in the hospital there was no fever, except on three brief occasions. The pulse varied between 80 and 90 beats per minute, and the respirations between 20 and 30. The blood pressure on September 22, 1930, was recorded as 140 systolic and 70 diastolic in the left arm and 135 systolic and 65 diastolic in the right. (It should be noted that although there was not a marked difference in blood pressure between the two arms in any of the various examinations, the right was constantly lower than the left.) The patient suffered from headache and vertigo. Recurrent ascites is mentioned, but no record of paracentesis abdominis is found. A right-sided pleural effusion developed, and was tapped twice with the removal of about a liter of fluid on each occasion. Dyspnea and anasarca increased gradually. Venesection gave temporary relief. On October 13, 1930, the patient died, probably of congestive heart failure.

Laboratory Examinations:

Urinalysis: amber color, acid reaction, specific gravity varied from 1.010 to 1.023, no sugar; albumin, a faint trace to a cloud; a few hyaline casts and a few leucocytes. There is no record of a blood count. The blood sugar was 100 mg. per cent; the blood urea nitrogen was 13 mg. per cent. The Wassermann reaction of the blood was strongly positive.

Electrocardiograms taken March 5 and May 15, 1930, showed left axis deviation and diphasic T-waves in Lead I.

Roentgenograms of the chest were taken on two different occasions. The first was on February 21, 1930, the day after his first admission (Fig. 7A). The report states "The cardiac shadow is slightly enlarged to the left. Over the ascending branch of the aorta there is a definite enlargement which, upon fluoroscopy, shows a definite expansile pulsation. There is considerable density over both bases. Diagnosis: Aneurysm of the ascending aorta." The second roentgenogram (Fig. 7B) was made on September 11, 1930. The report says, "There is general cardiac hypertrophy and considerable enlargement of the left ventricle. The silhouette would suggest aortic insufficiency. There is fluid at the right base obliterating the diaphragm. Lung fields show evidence of pulmonary congestion."

Necropsy was performed by Dr. Bucher and was reported as follows: "The pericardium is smooth and glistening. Pleural pericardial and peritoneal cavities contain no excess of fluid. The heart weighs 750 grams, and is markedly enlarged. Its muscles are thick, red and beefy. There is no coronary sclerosis. All of the valves are dilated, but there is no intrinsic valve defect. (There was no aortic valve disease to explain the aortic diastolic murmur.)[21] The first portion of the aorta is greatly enlarged and has assumed a fusiform spindle shape. There is a huge sac containing blood extending into the aorta and reaching down to the upper lumbar region. The aorta itself shows numerous contractions, great scarring and some atheromatous change."

Examination of the preserved specimen shows that the sac of the dissecting aneurysm began at the level of the aortic valve and encircled the right anterolateral two-thirds of the circumference of the ascending aorta. In this region there was a very large sacculation which bulged toward the right. This sacculation is in a position to be responsible for the bulge in the ascending aorta which is seen in the roentgenograms (Figs. 7A and 7B). The site of the primary rupture is not stated in the necropsy report, and it could not be ascertained from the preserved specimen. Whether or not the dissection extended along any of the large branches of the aorta can likewise not be determined. The aneurysm gradually tapered off and reëntered the aortic lumen in the upper lumbar region. Its walls were smooth and entirely lined with endothelium.

The lungs showed passive congestion. The spleen and kidneys were described as normal. The liver showed a nutmeg appearance and was much enlarged. Other organs showed nothing remarkable.

CASE XI. R. L., a white male, aged forty-one, was admitted to the Bryn Mawr Hospital September 6, 1928, on the service of Dr. John Sharp. He was known to have had a hypertension for some time, varying in the region of 200 systolic and 130 diastolic. He had experinced dyspnea on exertion and nocturia for some time and had been admitted to the hospital once previously (March 1 to June 5, 1928) for these symptoms. During the first week of August, 1928, he had an attack of acute abdominal pain, most marked in the right upper quadrant. A physician who saw him interpreted it as due to "congestion of the liver." The pain gradually subsided within a period of ten days, but the patient's general condition deteriorated. Then mental disturbances developed which necessitated his re-admission.

On examination there were no evidences of congestive failure. The only significant physical findings were a blood pressure of 210 systolic and 165 diastolic, slight arteriosclerosis and slight enlargement of the heart to the left. There was no dyspnea and no elevation of temperature or pulse rate.

During his stay in the hospital he gradually became worse, the main symptom being a depressive psychosis with occasional periods of irrationality. He gradually lost appetite, weight and strength, and died on January 11, 1929. The manner of his death was neither typically cardiac nor renal.

Laboratory Examinations:

There was no record of a blood count during this admission. One on March 18, 1928, showed erythrocytes 4,350,000, leucocytes 7,200, hemoglobin 76 per cent (Dare); differential: neutrophiles 74 per cent, lymphocytes 22 per cent, large mononuclears 4 per cent.

The urine showed amber color, alkaline reaction, 1.020 specific gravity, a light cloud of albumin, no sugar, two to three hyaline casts, a few leucocytes and no erythrocytes. Subsequent examinations showed varying amounts of albumin and casts, but no steady increase in the pathological elements in the urine.

The blood urea nitrogen (Sept. 7, 1928) was 20 mg. per cent.

There is no record of an eyeground examination.

A roentgen-ray examination of the chest during the first admission (April 30, 1928) showed slight widening of the aortic arch and moderate generalized cardiac enlargement. (The aneurysm probably developed during the first week of August, 1928, at the time of the attack of abdominal pain. The roentgen examination therefore probably antedated the formation of the dissecting aneurysm.)

A necropsy was performed on January 11, 1929, by Dr. W. P. Belk, two hours after death. The right lung showed well-developed bronchopneumonia and passive congestion. The kidneys showed chronic interstitial nephritis and a recent red infarct. The heart was greatly hypertrophied. The aorta showed many contracted scars in the thoracic portion. There were three enlargements around the aorta, in the region of the diaphragm, which on further examination proved to be dissecting aneurysms, partly filled with organized clots. The rupture in the aortic intima which had led to the formation of these aneurysms was situated just below the diaphragm. There was no external rupture. No mention is made of obstruction of any of the aortic branches by the aneurysm. Histological study of the aorta showed no evidences of syphilis.

The causes of death were given as (1) chronic interstitial nephritis, (2) multiple dissecting aneurysms of the aorta and (3) bronchopneumonia.

Case xii. F. D., a colored male, aged fifty-five, was admitted to the Philadelphia General Hospital, June 8, 1927, on the service of Dr. Loewenberg. He had at no time experienced an attack which might, in retrospect, have been considered to mark the onset of a dissecting aneurysm. He had apparently been well until April 1, 1927. Since then he had developed successively, dyspnea on exertion, edema of dependent parts, headache, and nocturia. These symptoms had progressed steadily and quite rapidly in severity since their onset. He had vomited once or twice shortly before admission. He had noticed a swelling in his scrotum since 1924, which had recently become considerably larger.

On admission the patient was confused mentally and mumbled his words. The temperature was 100°F., the pulse rate was 96, and respirations were 30 per minute. There were subconjunctival hemorrhages, and infraorbital edema. The blood pressure was not recorded. The heart showed enlargement, the apex being in the anterior axillary line. Heart sounds were faint, no murmurs were heard and the aortic second sound was of less intensity than the pulmonic second. There were evidences of marked congestive failure with extreme dyspnea and generalized anasarca. Over the bases of both lungs the percussion note was impaired, and many râles were heard. The liver extended one inch below the umbilicus. The scrotum was immense, extending to the level of the knees.

During his stay in the hospital the anasarca subsided considerably. The patient's mental state improved, but never entirely cleared up. The temperature which had been recorded as 100°F. on admission subsided to normal within a week, and remained at that level. The pulse rate dropped gradually to 80, but the respirations continued at a rate of from 25 to 30 throughout. On June 24, 1927, a systolic murmur was heard at the apex and base, and a short faint diastolic murmur was audible in the fourth left interspace close to the sternum "superficial and of a pericardial nature." The scrotum was tapped five times, and yielded between 1 and 2 liters of chocolate-colored fluid on each occasion. The last tapping was done on June 26, 1927. The final note on June 27 states: "Patient in good condition. Yesterday A.M., bled freely from hydrocele. Attended by Dr. Young who stopped the bleeding. In the afternoon, patient became quite weak and died at 5 P.M."

The following laboratory examinations were made: Urine analysis—amber color, acid reaction, specific gravity 1.020, a heavy cloud of albumin, no sugar, and no casts or blood cells. There was no record of a blood count. The Wassermann test of the blood was negative. The blood sugar was 104 mg. per cent. The blood urea nitrogen was 34 mg. per cent. No roentgenogram of the thorax was made.

Necropsy was performed on June 27 by B. A. Gouley and reported as follows: "The heart is enlarged, weighing 550 grams, the left ventricle is hypertrophied, the right, dilated. There is diffuse whitening of the pericardium over the anterior aspect of the right auricle and ventricle, not localized as in milk spots, but quite delicate and generalized. There is definite gross evidence of marked syphilitic aortitis. At the beginning of the transverse position of the arch there is an opening about 1.5 cm. in diameter which leads to a distinct and separate aortic

column. This unusual aneurysm is mildly dilated along the transverse and descending arches, and then runs down side by side with the original channel, being almost of the same caliber, to the bifurcation of the iliacs. It apparently started as a dissecting aneurysm at the transverse portion of the aortic arch. It is lined entirely by endothelium, and is fenestrated, the openings occurring in pairs at the intercostal levels, these fenestrations leading mostly to the original aorta. Certain parts of it are filled with laminated clots, which, however, do not completely occlude the channel. The syphilitic process has invaded the intima lining the aneurysm to a mild extent. This may not be a dissecting aneurysm but a pure anomaly." The patient may have died from hemorrhage into the scrotum, which extended upward and formed a hematoma at the brim of the pelvis and along the sheath of the left psoas muscle.

CASE XIII. A. C., a colored female, aged forty-six, was admitted to the Philadelphia General Hospital, July 27, 1927, on the service of Dr. Loewenberg. No accurate history could be obtained, but her husband stated that she had been complaining of "pains all over her body" during the past two months. For two weeks she had talked of pains in her left side; whether these were abdominal or thoracic could not be ascertained. She then became weak and "took to bed off and on." Four days before admission (July 23, 1930) she "went on a Saturday night party, took a few drinks," and was unable to get out of bed or to speak the next morning, remaining stuporous all day. On Monday she was slightly stronger, and got out of bed but never spoke. On Tuesday, she became weaker and more stuporous, vomited some blood during the evening, and had a profuse sweat.

On admission, Wednesday, July 27, the patient was described as gray-haired and obese. There was no edema. She was stuporous; she could be aroused, but did not answer questions. She was able to move all her extremities. The temperature was 100°F.; the pulse rate 100 per minute, and the respiratory rate, 30. The pupils were equal, regular, and sluggish in reaction; the left internal rectus muscle was weak. There were râles throughout both lungs. The blood pressure was 170 systolic and 95 diastolic. The heart was markedly enlarged, extending to the anterior axillary line on the left. The cardiac rhythm was regular. A systolic murmur was heard at the apex. The abdomen showed an indefinite "fecal"(?) mass, in the left lower quadrant. The patellar reflexes were absent. Babinski's sign was not present on either side.

The patient remained stuporous throughout her stay in the Hospital. Her temperature ranged between 100° and 103°F. The pulse varied from 100 to 130. The respirations were recorded as 30 to 40 per minute at various times. Shortly after admission she developed a right-sided hemiplegia. Despite hot packs, venesection, colonic irrigation and proctoclysis she became progressively more stuporous and died July 31, 1927, without a diagnosis having been established.

Laboratory reports: Urine (July 29) blood red, alkaline reaction, specific gravity 1.021, cloud of albumin, no sugar, many leucocytes, many erythrocytes. Blood count (July 29), erythrocytes 4,260,000, leucocytes 37,200, hemoglobin 83 per cent; differential: neutrophiles 72 per cent, lymphocytes 16 per cent, large mononuclears 12 per cent. The blood sugar was 113 mg. per cent. The blood urea nitrogen was 15 mg. per cent. The spinal fluid (July 28) showed no abnormal findings. The eyegrounds examination showed vascular sclerosis, silver wire arteries and a few areas of white exudate along certain of the vessels. No roentgenogram of the thorax was made.

Necropsy, performed July 31, was reported as follows: "The brain shows marked vascular sclerosis and thrombotic softening of multiple areas in the putamen, the right internal capsule, and in the left side of the pons. The heart weighs 400 grams, and is slightly enlarged but otherwise normal. There are a few scars, mostly atheromatous, in the root of the aorta. There is a dissecting aneurysm extending from the descending portion of the arch of the aorta to the bifurcation of the iliacs. About the level of the celiac axis there is a rupture of the intima and media, and from this point of origin there is extensive hemorrhage between the aortic layers." The report contains no further description of the lesion. "Diagnosis: cerebral thrombosis with multiple areas of softening; vascular lues; dissecting aneurysm of the aorta, sclerotic myocarditis, terminal bronchopneumonia, benign renal sclerosis, endocervicitis."

CASE XIV. E. W., a colored male, aged fifty-one, was admitted to the Philadelphia General Hospital, January 7, 1929, on the service of Dr. Strecker. He had been indefinitely ill for

three years but had been able to work until two weeks prior to admission when he had suffered a "stroke." He did not lose consciousness, but became stuporous, and unable to talk or move his limbs. Subsequently he had regained power in most of his members.

On admission he was mentally confused and drowsy and kept making slow aimless movements. There were no evidences of pain. The temperature was 96.6°F.; the pulse rate, 80; the respirations, 24 per minute. There were neurological signs of a past left-sided hemiplegia. The pupils were irregular, and their reaction to light was sluggish. There was a marked arcus senilis. The tongue moved normally, and showed tremor. No gag reflex was obtained, and deglutition was accomplished with difficulty. There were moderate venous pulsations in the neck. No abnormal physical signs were present over the lungs. Blood pressure was 185 systolic and 125 diastolic in the right arm, and 170 systolic and 130 diastolic in the left. The heart showed a forceful, diffuse apex beat in the 4th and 5th left interspaces, in the nipple line. The sounds were readily audible, and regular; no murmurs were heard. The peripheral arteries showed marked sclerosis. No other physical findings of note were recorded. A diagnosis of hypertension and partial left hemiplegia was made.

During the patient's stay in the Hospital, he became more stuporous, and his speech became slurred. There was no elevation of temperature, pulse or respiration at any time. On February 25, the final note was made, "The patient died suddenly and unexpectedly. He appeared unchanged 2/24/29 P.M. and was mumbling to interne, but died a few hours later. Never cleared mentally—death probably cardiac."

The laboratory tests were reported as follows: Urine, amber color, alkaline reaction, 1.020 specific gravity, faint trace of albumin, no sugar, many hyaline casts, many leucocytes. There is no record of a blood count. The Wassermann test of the blood was negative on three occasions. The blood sugar was 105 mg. per cent. The blood urea nitrogen was 28 mg. per cent. The spinal fluid showed no cells, no globulin, and a negative Wassermann reaction and colloidal gold curve. A roentgenogram of the thorax was not made.

Necropsy, performed by Dr. Lucke on February 25, was reported as follows: "The pericardium is distended with 80 c.c. of partly clotted blood. The heart is enlarged, weighing 540 grams. The epicardium is normal. The coronary arteries are somewhat stiff and tortuous, and show atheromatous changes. The posterior wall of the ascending aorta is discolored and bulges slightly; there is a small ragged tear through which blood has escaped into the pericardiac sac, the tear being located about 3 cm. above the aortic ring. There is a dissecting aneurysm which encircles the aorta to about the junction of the thoracic and abdominal portions. The mass of blood within the aortic walls is partly fluid and partly clotted, and varies in thickness from a few millimeters to 1 cm. About 3 cm. below the orifice of the left subclavian artery there is a small ragged tear in the inner wall of the aneurysm, probably due to an atheromatous ulcer. A similar tear is situated 2 cm. below the upper one. Through these ruptures the blood has forced its way between the aortic layers. Subsequently the dissecting aneurysm has ruptured into the pericardium and caused death by compression of the heart. The intima of the aorta showed a relatively small number of hyaline and atheromatous plaques, and in the ascending portion it has a practically smooth and normal surface. The lungs show congestion. The kidneys are normal. The brain shows marked arteriosclerosis of all its vessels, large and small, with multiple areas of thrombotic softening."

Examination of the preserved specimen showed that the dissecting aneurysm surrounded one-half to two-thirds of the aortic circumference. There was no rëentrance of the blood into the aortic lumen. There was a marked sacculation of the aneurysm in the region of the junction of the transverse and descending portions of the aortic arch.

Case xv. C. B., a white male, aged sixty-seven, was admitted to the Philadelphia General Hospital, July 7, 1921, on the service of Dr. Piersol. He had had a "stroke" in 1916 which produced muscular paralysis (side not specified), and derangement in mind and speech. He partially recovered from this accident, only to be permanently invalided by another similar one in 1917. He had never walked since that time.

He was described on admission as a large, confused, white male with unintelligible speech. The temperature was 99°F., the pulse rate 80, and the respiratory rate 25. There was evidence of residual neurological change from an old right-sided hemiplegia. The pupils were slightly irregular and sluggish in reaction. The blood

pressure was 190 systolic and 90 diastolic. Examination of the lungs showed emphysema. The heart was described as normal in size and position. There were no other significant physical findings recorded.

His stay in the hospital was uneventful, from our point of view, until September 4, 1921. On this day he was apparently in status quo, and sitting in a chair at noon, eating his dinner. At 1.50 P.M. he was found dead in his chair by a nurse.

Laboratory studies: Urine analysis—clear, acid reaction, specific gravity 1.026, a trace of albumin, no sugar, a few granular casts, occasional leucocytes. There was no record of a blood count. The blood urea nitrogen was 29 mg. per cent. The blood chlorides were 561 mg. per cent. Blood creatinine was 0.6 mg. per cent.

A necropsy, performed September 4, showed a rupture of the aortic intima just above the semilunar valve. A dissecting aneurysm extended from this point up to the origin of the great vessels of the neck. There was clotted blood in the aneurysm, and around the pulmonary artery. There was no evidence of organization. The dissecting aneurysm had ruptured downward into the pericardial sac, which contained several ounces of freshly clotted blood. The lumen of the aortic arch was somewhat widened. The intima showed "a fair number of hyaline plaques and a few insignificant scars." The innominate artery was absent, the right common carotid and right subclavian arteries arising independently from the aorta. Both of these vessels were considerably dilated at their origins, and their intimal linings showed numerous depressed scars and puckers. The heart was hypertrophied, weighing 500 grams. There was no demonstrable endocardial lesion. The coronary vessels were stiff and atheromatous. There was generalized arteriosclerosis, which was particularly apparent in the renal and cerebral vessels.

In this case it is quite probable that there was only a brief interval between the initial intimal rupture and death from rupture into the pericardium.

CASE XVI. M. D., a colored female, aged forty-four, was admitted to the Philadelphia General Hospital May 8, 1930, on the service of Dr. Lull. The patient was in a semicomatose state, and no history could be obtained except that she had felt weak and sick for about a week. She did not know how many children she had had. Her last menstrual period had been on September 3, 1929.

The patient was described as a well-nourished negress who was stuporous and answered questions vaguely. Her eyes rolled upwards. There were no muscular twitchings. The neck, chest, and heart were described as showing no abnormal physical signs. The blood pressure was 240 systolic and 130 diastolic. The abdominal examination showed a pregnant uterus whose fundus extended above the umbilicus. Fetal heart sounds were heard in the left lower quadrant. The head was not engaged. The cervix was not dilated. The extremities were negative.

A diagnosis of pregnancy, eighth month, and toxemia of pregnancy was made. The patient was actively treated by venesection, spinal puncture, colonic irrigation, hypodermoclysis and intravenous saline and glucose. She improved slightly under this regimen. On May 12, 1930, she went into labor and delivered herself spontaneously at 7.50 A.M. of a female child, in whom considerable stimulation was necessary to establish respiration. After delivery the mother's condition was described as poor. That afternoon she complained of "pain in the back and abdomen," and was restless. She gradually subsided into coma and died on May 15, 1930, at 2.15 P.M.

During her sojourn in the hospital the patient's temperature was normal except for a slight post-partum rise to 100°F. The pulse rate varied from 20 to 25 per minute. Urinalysis on admission showed amber color, acid reaction, specific gravity 1.008, a heavy cloud of albumin, no sugar, and many hyaline and granular casts. Another examination on May 13 showed no albumin and no casts. Blood urea nitrogen on admission was 45 mg. per cent. Blood sugar was 82 mg. per cent. No other special studies were reported.

Necropsy was performed on May 15 by Dr. Zwecker. "The pleural and peritoneal surfaces show no significant findings. On opening the pericardium a large mass of clotted blood is found over the heart. It appears to be a recent extravasation. The heart weighs 510 grams; the left ventricle is greatly hypertrophied. The valves are normal. The aorta is the seat of a dissecting aneurysm. The initial intimal rupture is in the anterior wall of the ascending aorta, 1 cm. above the semilunar valves. It is a sharp transverse cut involving half the aortic circumference. The dissection, starting from this

point completely encircles the ascending aorta, and then tapers off gradually. It extends from the aortic valve to the bifurcation of the iliacs and into the femoral arteries. It extends out along all the large branches of the aortic arch. A small amount of blood has escaped into the soft tissues of the neck, just above the clavicle, and a slight extravasation extends downwards into the anterior mediastinum. Clots of blood are found in the cavity of the aneurysm. An organized thrombus is present in that part of the sac which surrounds the root of the aorta. The external wall of the aneurysm shows localized dilatations here and there along its entire extent. The final rupture into the pericardium took place through a small jagged opening in the outer wall of the aneurysm on the right side of the ascending aorta about 1 cm. higher than the initial intimal tear. At this point there is a localized weakening and dilatation. The aorta, particularly in its lower half, shows many elevated areas of intimal fibrosis and fatty changes. There is no calcification. The lungs, spleen, liver, gastrointestinal tract and pancreas show no abnormalities. The kidneys show fairly marked arteriolar nephrosclerosis. The uterus shows the expected post-partum changes."

One cannot be sure just when this dissecting aneurysm developed. The most likely time was before admission. It probably did not occur during delivery, since there was an organized thrombus in the lumen of the aneurysm at one point.

Case xvii. (*Case of non-fatal perforation of a syphilitic saccular aneurysm with mediastinal hemorrhagic extravasation shown by roentgenogram.*)

M. D., a colored male, aged twenty-nine, was admitted to the Philadelphia General Hospital on the service of Dr. Loewenberg, October 12, 1929. He had suffered from pain in the left side of the back for ten months; there was an additional history of post-prandial epigastric discomfort. The chief findings on physical examination were: blood pressure 130 systolic and 75 diastolic; a blowing systolic murmur at the aortic area; a decreased motility of the left chest; absent fremitus, vocal resonance and breath sounds, and a dull percussion note, over the lower part of the left chest; a pulsating mass in the epigastrium, over which a murmur was heard; and tenderness in the epigastrium and over the left lower chest.

Roentgenographic examination (Fig. 8) showed an erosion of the bodies of the 10th, 11th and 12th thoracic vertebrae, and of the 11th rib on the left side; a somewhat diffuse shadow in the left chest; a denser shadow extending upward and to the left from the region of the aortic knob; a displacement of the heart and trachea to the right, and a shadow to the right of the right cardiac border.

On October 14, 1929, the patient grew suddenly weak and died.

Necropsy performed by Dr. Stewart on October 14, 1929, revealed a ruptured saccular aneurysm of the aorta, just below the diaphragm.

One or more non-fatal perforations had apparently occurred before the final one, because there was a considerable amount of firmly clotted, and possibly organized blood in the mediastinum, and in the tissues behind the left pleura. These hemorrhagic extravasations were probably the cause of the shadows in the mediastinum and left lung field, which appear in the roentgenogram. The final, fatal rupture had taken place into both the mediastinal tissues and the left pleural cavity. Erosion of the lower four thoracic and 1st lumbar vertebrae was found. Definite evidence of syphilitic aortitis was present.

We wish to thank Dr. Mabel Gates, Dr. Vincent Patrick, Dr. F. J. Hirschboeck, Dr. C. J. Tidmarsh, Dr. H. E. MacDermot, Dr. E. H. Goodman, and Dr. E. M. Hall for their kindness in sending us information concerning their cases, and for permission to publish this information.

We wish to acknowledge the kindness of Dr. Alfred Stengel, Dr. Allyn, Dr. Loewenberg, Dr. Lull, Dr. John S. Sharp, Dr. Strecker and Dr. Piersol in allowing us to report cases from their services.

REFERENCES

1. Adami, J. G. Upon arrested or repaired dissecting aneurysms, with reports of 2 cases. *Montreal M. J.*, 1895–1896, *24*, 945–954; 1896–1897, *25*, 23–36.
2. Babes, V., and Mironescu, T. Ueber disseziererende Arteriitis und Aneurysma dissecans. *Beitr. z. path. Anat. u. z. allg. Path.*, 1910, *48*, 221–235.
3. Bostroem, E. Das geheilte Aneurysma dissecans *Deutsches Arch. f. klin. Med.*, 1887, *42*, 1–74.
4. Crowell, P. D. Dissecting aneurysms of the

aorta; report of cases and review of literature. *J. Am. M. Ass.*, 1921, *77*, 2114.

5. Davy, H., and Gates, M. A case of dissecting aneurysm of the aorta. *Brit. M. J.*, 1922, *1*, 471–472.
6. Duftschmid, E. Ueber einem Fall von Aneurysma dissecans Aortae mit Lahmungseischeinungen. *Wien. klin. Wchnschr.*, 1925, *38*, 119–123.
7. Dumas, A., Croizat, P., and Lyonnet, R. Anevrysme disséquant, en "canon de fusil" de l'aorte thoracico-abdominale. Crise solaire terminale. *Lyon méd.*, 1929, *143*, 394–398.
8. Erb, W., Jr. Experimentelle und histologische Studien über Arterienerkrankung nach Adrenalininjektionen. *Arch. f. exper. Path. u. Pharmakol.*, 1905, *53*, 173–212.
9. Finny, M. *Lancet*, 1885, *2*, 69.
10. Fischer, B. Die experimentelle Erzeugung vom Aneurysmen. *Deutsche med. Wchnschr.*, 1905, *31*, 1713–1716.
11. Fisher, N. F. Changes at the orifices of the intercostal arteries in a dissecting aneurysm of the aorta. *Tr. Chicago Path. Soc.*, 1927, *12*, 351.
12. Frei, C. *Inaug. Diss.*, 1921. Quoted by Loeschke, ref. 29.
13. Freistadt, K. Zur Symptomatologie und pathologischen Anatomie des Aneurysma dissecans. *Virchow's Arch. f. path. Anat. (etc.)*, 1922, *237*, 63–74.
14. Gager, L. T. The symptoms of dissecting aneurysm of the aorta. *Ann. Int. Med.*, 1929, *2*, 658–664.
15. Gilchrist, A. R., and Ritchie, W. T. The ventricular complexes in myocardial infarction and fibrosis. *Quart. J. M.*, 1930, *23*, 273–292.
16. Goddard, P. B. Account of a case of dissecting aneurysm seen at an early stage. *Am. J. M. Sc.*, 1839, *23*, 20–21.
17. Goodman, F. H. Hemothorax caused by rupture of a dissecting aneurysm. Recovery. *J. Am. M. Ass.*, 1914, *62*, 1242–1244.
18. Griffith, K. S. Coarctation of the aorta. Paper read before Section on General Medicine, College of Physicians, Philadelphia, Dec. 22, 1930.
19. Hall, E. M. Healed dissecting aneurysm of the aorta. *Arch. Path. & Lab. Med.*, 1926, *2*, 41–49.
20. Hanser, A. Vom Aneurysma dissecans der Aorta zugleich über eine neue Entstehungsweise der Hämoglobinurie. *Deutsches Arch. f. klin. Med.*, 1926, *152*, 61–81.
21. Hart, C. Ueber einen Fall von "geheiltem" Aneurysma dissecans der ganzen Absteigenden Aorta bei einem Kriegsteilnehmer. *Berl. klin. Wchnschr.*, 1917, *54*, 693–695.
22. Hirschboeck, F. J., and Boman, P. G. A case report of dissecting aneurysm of the aorta, with distinctive x-rays findings. *Minnesota Med.*, 1922, *5*, 724–726.
23. Howard, C. P. Aortic insufficiency due to rupture by strain of a normal aortic valve. *Canad. M. Ass. J.*, 1928, *19*, 12–24.
24. Hubschmann. Schneidenformiges Aneurysma dissecans der Aorta. *München. med. Wchnschr.*, 1916, *1*, 394.
25. Krukenberg, E. Beiträge zur Frage des Aneurysma dissecans. *Beitr. z. path. Anat. u. z. allg. Path.*, 1920, *57*, 329–351.
26. Külbs. Experimentelle Studien über die Wirkung des Nebennierenextraktes. *Arch. f. exper. Path. u. Pharmakol.*, 1905, *53*, 140–156.
27. Lampert, H., and Müller, W. Bei welchem Druck kommt es zu einer Ruptur der Gehirngefässe? *Frankfurt. Ztschr. f. Path.*, 1925–1926, *33*, 471–477.
28. Latham and Swaine. Case of dissecting aneurysm of the aorta. *Tr. Path. Soc. Lond.*, 1856, *7*, 106.
29. Loeschke, A. Aneurysma dissecans auf luetischer Grundlage. *Frankfurter Ztschr. f. Path.*, 1928, *36*, 56–81.
30. Lundberg, A. Three cases of healed aortic rupture. *Acta med. Scandinav.*, 1930, *73*, 19–44.
31. MacCallum, W. G. Dissecting aneurysm. *Johns Hopkins Hosp. Bull.*, 1909, *20*, 9–12.
32. MacDermot, H. E. Four cases of aneurysm. *Canad. M. Ass. J.*, 1930, *23*, 248–251.
33. Mager, W. Ein Beitrag zur Lehre von den Erkrankungen der Aorta. *Ztschr. f. Heilk.*, 1903, *24*, 323–333.
34. Moosberger, W. Zur Symptomatologie des Aneurysma dissecans. *Schweiz. med. Wchnschr.*, 1924, *54*, 325–330.
35. Morgagni, J. B. The Seats and Causes of Diseases Investigated by Anatomy. In five books containing a great variety of dissections, with remarks. Translated from the Latin by Benjamin Alexander. A. Millar and T. Cadell, London, 1769. Vol. 1, p. 802.
36. Moriani, G. Ueber einen Fall von Aneurysma dissecans aortae mit besonderer Berücksichtigung der frischer Rupturen der Aortenmedia. *Virchow's Arch. f. path. Anat. (etc.)*, 1910, *202*, 283–305.
37. Neuberger, J. Zwei Fälle von syphilitischem Aneurysma der Arteria pulmonalis. *Deutsche med. Wchnschr.*, 1930, *56*, 821–823.
38. Osler, W. Modern Medicine. Lea Brothers & Co., Philadelphia, 1908, *4*, 468.
39. Osler, W., and McCrae, T. Modern Medicine. Lea and Febiger, Philadelphia, 1915, *4*, 467.
40. Paschkis, K. Ueber Aortenruptur bei intakter Gefässwand. *Med. Klin.*, 1925, *21*, 1921.
41. Patrick, C. V., and Taylor, J. F. A case of dissecting aneurysm of the aorta. *Lancet*, 1929, *1*, 181–182.

42. Peacock. Report on cases of dissecting aneurysm. *Tr. Path. Soc. Lond.*, 1863, *14*, 87.
43. Pennock, C. W. Case of anomalous aneurism of the aorta resulting from effusion of blood between the laminae composing the middle coat of that vessel. *Am. J. M. Sc.*, 1839, *23*, 13–19.
44. Reitter, K. Aneurysma dissecans und Paraplegia, zugleich ein Beitrag zur Pathologie der Blutzirkulation im Rückenmark. *Deutsches Arch. f. klin. Med.*, 1916, *119*, 561.
45. Resnik, W. H., and Keefer, C. S. Dissecting aneurysm with signs of aortic insufficiency; report of a case in which the aortic valves were normal. *J. Am. M. Ass.*, 1925, *85*, 422–424.
46. Schede, F. Beiträge zur Aetiologie, Verlauf und Heilung des Aneurysma dissecans der Aorta. *Virchow's Arch. f. path. Anat.* (*etc.*), 1908, *192*, 52–86.
47. Schilling, F. Ueber Aneurysma dissecans der Aorta. *Frankfurt. Ztschr. f. Path.*, 1922, *27*, 336–352.
48. Schmidt, E. *Inaug. Diss.*, 1911. Quoted by Loeschke, ref. 29.
49. Sella, H. Aortenruptur und Aortenaneurysma bei Aortenstenose am Isthmus. *Beitr. z. path Anat. u. z. allg. Path.*, 1910, *49*, 501–528.
50. Singer, R. Experimentelle Studien über die Schmerzempfindlichkeit des Herzens und der grossen Gefässe und ihre Beziehung zur Angina pectoris. *Wien. Arch. f. inn. Med.*, 1926, *12*, 193–208; 1926–1927, *13*, 157–178; 1927, *14*, 113–128.
51. Sutton, D. C., and Lueth, H. C. Pain. *Arch. Int. Med.*, 1930, *45*, 827–867.
52. Tidmarsh, C. J. Rupture of the aorta. *Canad. M. Ass. J.*, 1930, *23*, 416–417.
53. von Schnurbein, F. Ueber Aortenruptur und Aneurysma dissecans. *Frankfurt. Ztschr. f. Path.*, 1926, *34*, 532–561.
54. Walker, C., and Walker, L. Sudden detachment of aortic intima (so-called dissecting aneurysm). *Brit. M. J.*, 1919, *2*, 200.
55. Whitman, R. G., and Stein, H. B. A contribution to the pathogenesis of dissecting aneurysms; a case of dissecting mesaortitis (Babes and Mironescu), without dissecting aneurysm. *J. Med. Research*, 1924, *44*, 579–592.
56. Wys, O. Aneurysma dissecans der Aorta ascendens. *Arch. d. Heilk.*, 1869, *10*, 490.

THE DIFFERENTIAL DIAGNOSIS OF ORGANIC HEART DISEASE BY THE ROENTGEN RAY*

By GEORGE LEVENE, M.D., *and* WILLIAM D. REID, M.D.
BOSTON, MASSACHUSETTS

THE value of the roentgen ray in the diagnosis of diseases of the heart appears to be a variable factor. The aid deriving from its application to the routine study of the heart is not generally conceded; its ability to assist in a differentiation of the factors operable in disturbances of the heart is not generally sought.

While the work of Vaquez[11] and Vaquez and Bordet,[10] Köhler,[6] Holmes,[4] Assmann[1] and others, has tended to establish the roentgen ray as an important adjunct in the study of the heart, many are as yet content with a mere indication for its use in the detection of cardiac enlargement.

Our study was begun with the purpose of evaluating the roentgen ray in the diagnosis of suspected disturbances of the heart. While this agent must be regarded only as a complement to the valuable clinical and laboratory methods generally employed, it was thought best to make the conditions of our investigation as exacting as possible. It was therefore decided that we would attempt to derive our conclusions from the roentgenoscopic examination and film, alone, unassisted by any information ordinarily necessary for cardiac diagnosis. In other words, it was desired to ascertain just how far the roentgen findings alone would go in establishing a diagnosis.

While erroneous deductions were occasionally derived, the value of the method, in our hands, was encouragingly demonstrated. With increasing experience we learned to obtain from roentgenoscopic examination important information regarding the functional states of the heart; the film was more useful in portraying gross, structural changes of the various chambers.

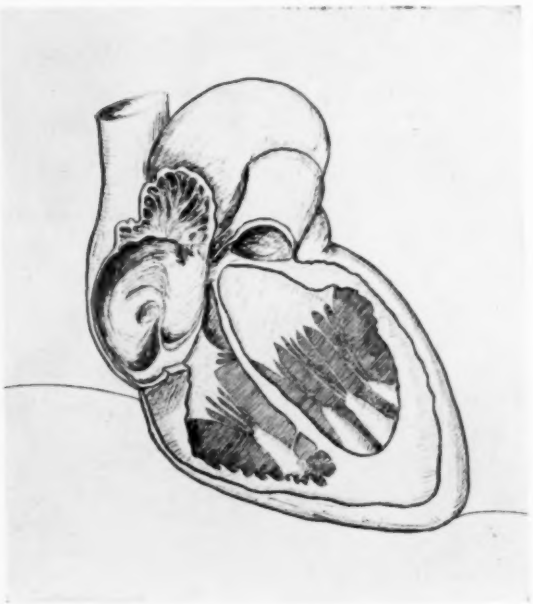


FIG. 1. Semi-diagrammatic illustration of the heart, showing the topography of the chambers and great vessels (see Fig. 2).

HEART MEASUREMENTS

Measurements of the heart suggested by Vaquez and Vaquez and Bordet, many of which do not appear to be commonly in use, were of great assistance in the differentiation of heart lesions, and, in some instances, enabled us to detect lesions in hearts which were not enlarged, and which might otherwise very readily be regarded as normal.

While the roentgenogram actually shows only the outline of the heart, the latter presents certain fixed landmarks which enable us to determine the development of its several cavities (Fig. 1). The measurements which were derived by Vaquez and Bordet (Table 1) on the basis of the extensive anatomical researches of Rummo,[9] are as follows:

* From the Departments of Roentgenology and Cardiology of the Evans Memorial for Clinical Research and Preventive Medicine, Boston.

TABLE I

TABLE OF CARDIAC MEASUREMENTS (MODIFIED AFTER VAQUEZ AND BORDET)

Lesion		Length	Transverse	Oblique	Left Ventricle	Bisector	Right Ventricle	Left Auricle	Right Auricle	Index of Auriculo-Ventricular Ratio
Normal	Maximum	15.0	14.5	10.5	8.5	2.0	14.7	5.0	6.5	0.704
	Minimum	11.5	9.5	9.3	6.7	0.6	8.5	3.5	3.5	0.534
Mitral stenosis	Maximum	15.6	15.4	13.7	7.0	1.0	14.5	7.5	8.0	1.000
	Minimum	12.0	10.5	9.0	4.3	0.2	10.5	5.0	5.4	0.750
Mitral insufficiency	Maximum	16.8	16.0	12.5	9.2	1.7	15.3	7.0	7.7	0.600
	Minimum	13.7	13.7	11.8	7.0	0.5	13.0	4.0	6.0	0.400
Double mitral	Maximum	21.0	21.0	17.0	9.5	2.0	18.9	8.2	11.5	0.800
	Minimum	15.2	13.7	13.3	6.2	0.5	14.0	5.8	6.5	0.534
Aortic stenosis	Maximum	17.5	16.8	12.8	10.8	3.5	16.0	7.8	8.0	0.500
	Minimum	15.0	14.0	11.0	9.5	1.2	13.5	4.5	6.5	0.300
Aortic insufficiency	Maximum	20.0	19.6	15.5	13.2	3.5	19.0	7.0	8.0	0.500
	Minimum	12.7	13.0	10.5	8.5	1.0	12.7	3.2	5.0	0.300
Double aortic	Maximum	20.5	19.3	15.8	14.5	3.0	18.4	7.5	7.0	0.500
	Minimum	15.7	15.5	11.4	9.7	1.5	14.3	4.0	5.5	0.300
Hypertension	Maximum	17.0	15.5	12.5	10.0	2.5	15.5	6.5	7.5	0.650
	Minimum	12.5	12.0	10.0	8.0	1.3	13.0	5.0	5.8	0.400
Hyperthyroid	Maximum	15.0	14.5	10.5	8.5	1.5	14.7	7.0	6.7	0.704
	Minimum	11.5	9.5	9.3	6.7	0.5	8.5	4.0	4.0	0.534
Hypothyroid	Maximum	20.5	19.0	12.6	10.2	2.4	17.6	6.0	7.8	0.704
	Minimum	12.0	14.4	11.1	8.1	1.2	10.2	4.2	4.2	0.534
Pulmonary stenosis	Maximum	16.0	15.5	14.0	8.5	2.0	16.0	6.0	8.0	0.704
	Minimum	13.5	10.0	9.5	7.0	0.6	10.0	5.0	5.0	0.500

A line drawn through the center of the spinous processes serves as a mid-line (Fig. 2). Perpendiculars erected to the mid-line from the point of greatest width of the heart shadow on the right, and on the left, give the right and left median diameters. The left is usually twice as long as the right. According to Dietlen,[2] the ratio is, in man, standing, 1:1.9; reclining, 1:2.1; in woman, reclining, 1:2.4. Their combined length is usually not over 50–60 per cent of the pulmonary field at its greatest width, establishing the so-called "cardio-thoracic ratio." The median diameters correspond approximately to the width of the heart as determined by clinical percussion.

The apex of the heart is usually situated at the point where the cardiac outline crosses the left diaphragm, though in occasional enlargements its position may vary, and its exact localization be obtained only by roentgenoscopic examination.

A line drawn from the apex to the junction of the right auricle and vascular arc determines the length of the heart. The angle formed by this line and the mid-line shows the degree of rotation of the heart on its vertical axis. The importance of this

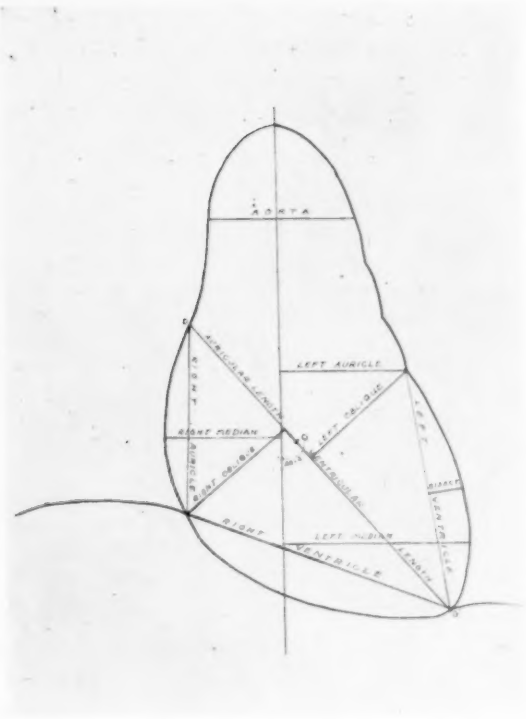


FIG. 2. Diagram showing cardiac measurements. The points of measurement are derived from anatomical landmarks (see Fig. 1).

measurement may be understood when it is recalled that the greater the angle, the farther out will be the apex, and thus, the larger the area of cardiac dullness. As in obesity, ascites, etc., the heart is pushed up, the angle becomes greater; and, while the transverse diameter of the heart may exceed its normal value, measurement of the axis shows this to be due to displacement, rather than enlargement. In the sthenic individual this angle varies between 50 and 70°; in the asthenic type it ranges between 45 and 50°.

A line dropped perpendicularly to the length of the heart, from the junction of the left auricle and left ventricle, and from the cardio-hepatic angle, establishes, respectively, the left and right oblique diameters. These correspond more nearly to the width of the heart at its base.

The roentgenographic silhouette of the heart being composed of auricles and ventricles, it is useful to know how much of the total area is due to each. An index of auriculo-ventricular ratio is thus obtained by bisecting the length of the heart by a line joining the cardio-hepatic angle on the right, and the auriculo-ventricular junction on the left (Fig. 2). The value of the upper segment (*OD*) is divided by the value of the lower segment (*OG'*), and, in normal hearts, is found to vary between 0.534-0.704. In conditions producing a preponderant enlargement of the ventricles, the index falls to 0.400–0.200. In conditions producing a preponderant enlargement of the auricles, the index rises to 0.800–0.900, or even 1.000. The index of auriculo-ventricular ratio, as will be shown, is often of great value in establishing a differentiation of various lesions, and in some instances points to a lesion in the absence of other roentgenological evidence.

A line joining the apex with the auriculo-ventricular junction on the left, reveals the size of the left ventricle, and a perpendicular to this line erected at the point of greatest salience of the ventricular curve, establishes a bisector of its arc, with a value equivalent to the development of the left ventricular myocardium. In ventricular hypertrophy the bisector is large.

A perpendicular to the mid-line from the auriculo-ventricular junction shows the extent of the left auricle, and its length varies with the size of the chamber.

A line joining the cardio-hepatic angle and the apex measures the size of the right ventricle.

A line joining the point of junction of the right auricle and vascular arc with the cardio-hepatic angle shows the development of the right auricle.

It thus becomes possible, on the basis of these measurements, to establish to what extent each chamber of the heart enters into a particular picture of cardiac enlargement. It is apparent that a prominent right auricle may not be an enlarged auricle; rather, it may represent displacement to the right by an enlarged right ventricle. While the right median diameter is of comparatively little value in determining this

question, measurements of the respective cavities are of definite assistance.

Roentgenoscopic examination in the various oblique and lateral positions is made of all cases, and films in these positions obtained when indicated. The barium meal is frequently administered as an aid in the interpretation of enlargement or displacement which often produces pressure deformities of the esophagus.

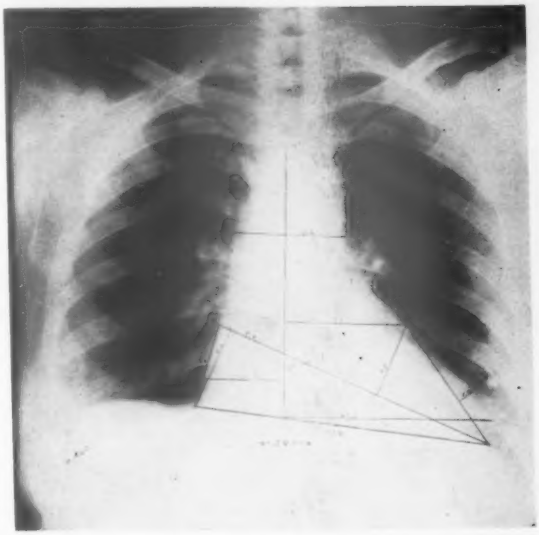


Fig. 4. Mitral insufficiency. The left median diameter shows greatest change due to enlargement of the left ventricle and lateral displacement of the apex by an enlarged right ventricle. The low index of auriculo-ventricular ratio shows preponderant ventricular enlargement.

VALVULAR LESIONS

Distinctive cardiopathies usually produce characteristic alterations in the form of the heart. A consideration of the gross structural changes is therefore essential to an interpretation of the roentgenographic silhouette.

It is appreciated that the exact reason for hypertrophy of the heart is not as yet conclusively established, and that the conception of "back-pressure" is open to question. However, since the latter has obtained wide acceptance, it was utilized as a working hypothesis in these studies. It becomes of interest to observe to what extent, if any, the use of the "back-pressure" conception has led to erroneous conclusions in this series. The theory advanced by Mackenzie[7] that heart failure is dependent upon lack of forward propulsion does not lend itself so readily to the analysis of roentgen findings in the diagnosis of heart disease.

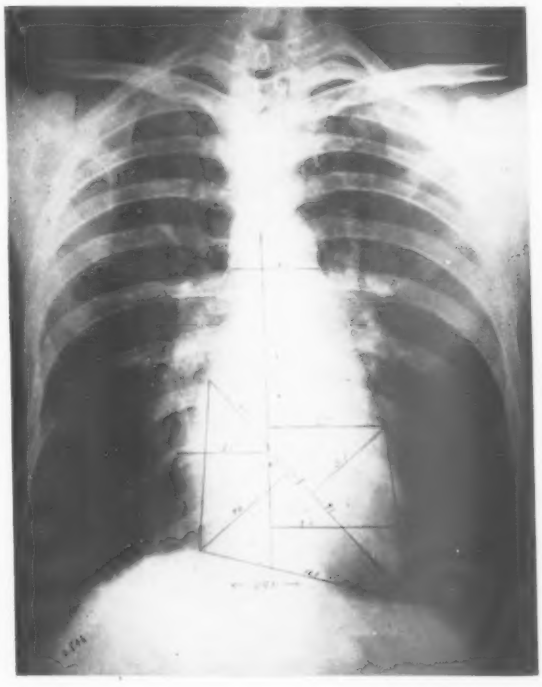


Fig. 3. Mitral stenosis. Transverse diameter of heart 13.1 cm., pulmonary field 29.0 cm., apparently not enlarged. Note the small ventricles and comparatively large auricles, with index of auriculo-ventricular ratio of 0.846 which is typical of this lesion.

MITRAL STENOSIS

The pathology of mitral stenosis consists of a narrowing of the mitral orifice. This results in an increase of pressure within the left auricle, which, being comparatively thin-walled, dilates. At the same time, this pressure is transmitted through the valveless pulmonary veins to the lungs, which become congested ("brown induration"). An increased pressure is thus encountered by the right side of the heart, and it is transmitted through the right ventricle to the right auricle. The right auricle, also thin-walled, dilates. In consequence of a

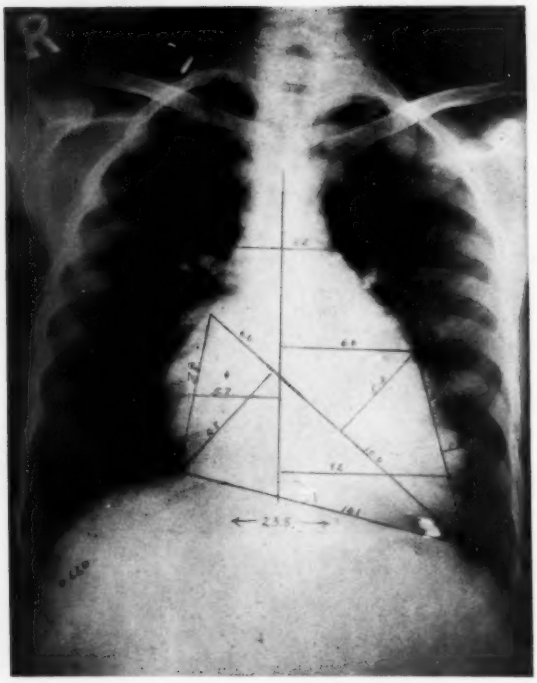


Fig. 5. Mitral stenosis and insufficiency—the typical "mitral heart." Note how this combines the characteristics of the two lesions (see Figs. 3 and 4).

diminished flow of blood to the left ventricle, the latter does not enlarge. Not infrequently its size becomes reduced.[5] The changes in contour therefore consist of enlarged auricles and practically normal-sized ventricles. Early mitral stenosis may therefore be easily overlooked on the roentgenogram because of the slight changes in contour of the heart. We have found a valuable aid in the diagnosis of this condition to be the index of auriculo-ventricular ratio. In mitral stenosis it may range from 0.750–1.000. The typical picture, however, includes enlargement of both auricles (Fig. 3). This is shown on the film by an increase in the values of right median, right auricular and left auricular diameters. Not infrequently, the ventricles, or the entire heart, are not enlarged. The tendency to associate heart disease with enlargement of that organ, and to regard as enlarged only those hearts whose transverse diameter exceeds one-half the measurement of the pulmonary field would therefore be responsible for the failure to recognize this lesion.

MITRAL INSUFFICIENCY

Mitral regurgitation presents an incompetent mitral valve which allows a backflow of blood into the left auricle during ventricular systole. The auricle therefore increases in size. During contraction, the auricle passes more than its normal amount of blood into the left ventricle. This results in an increase in size of the left ventricle, together with a thickening of its walls, which rounds out its lateral border and apex. Increased pulmonary resistance encountered by the right ventricle causes the latter to undergo appreciable increase in size. Thus, the changes which affect the contour of the heart are: increased size of left auricle, increased size of left ventricle, with rounding of its lateral arc and apex, and increased size of right ventricle. These findings are demonstrated by an increasing value for the left auricular and ventricular cavities, increase in transverse diameter of

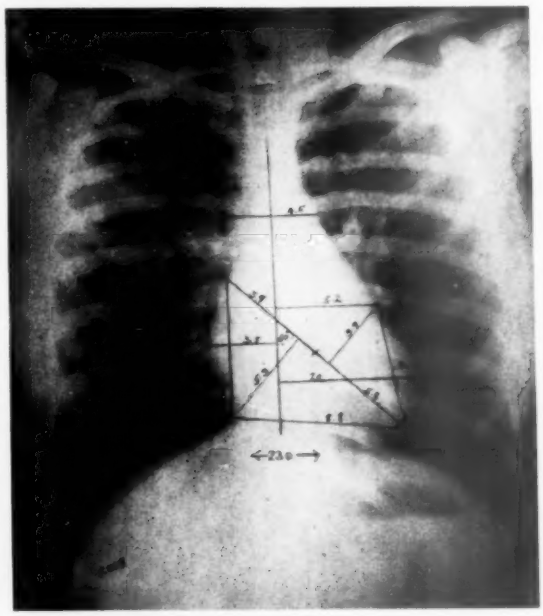


Fig. 6. Mitral stenosis with beginning insufficiency (child aged twelve). Transverse diameter of heart 10.5 cm., pulmonary field 23.0 cm., showing uncertainty of the "cardio-thoracic ratio" as a diagnostic criterion. Note the prominent auricles and high index of auriculo-ventricular ratio typical of mitral stenosis and the rounding and elevation of the apex due to insufficiency.

the heart, increase in the size of the right ventricle, and an index of auriculo-ventricular ratio of less than 0.500, which shows preponderant ventricular enlargement (Fig. 4). The enlargement is evidenced more in the transverse diameter than in the length of the heart.

DOUBLE MITRAL LESIONS

Mitral stenosis and insufficiency in the same heart, combining the pathological characteristics of the two lesions, present a roentgenographic silhouette which reveals the typical conformation due to each factor. The typical "mitral heart" therefore shows an increase in size of both auricles and ventricles (Fig. 5). These changes are shown by an increase in right and left median diameters, and, more particularly, by the increased measurement of the respective cavities. While the index of auriculo-ventricular ratio may be within normal limits, it may tend toward high or low values depending upon whether stenosis or insufficiency predominates. An interesting possibility of observing the progress of mitral heart disease is thus afforded by the index of auriculo-ventricular ratio (Fig. 6). Mitral insufficiency supervening upon a previous mitral stenosis reduces the value of the originally high index; whereas the latter becomes increased when mitral stenosis follows upon mitral insufficiency. These changes seem to occur earlier than recognizable alterations of cardiac contour and size.

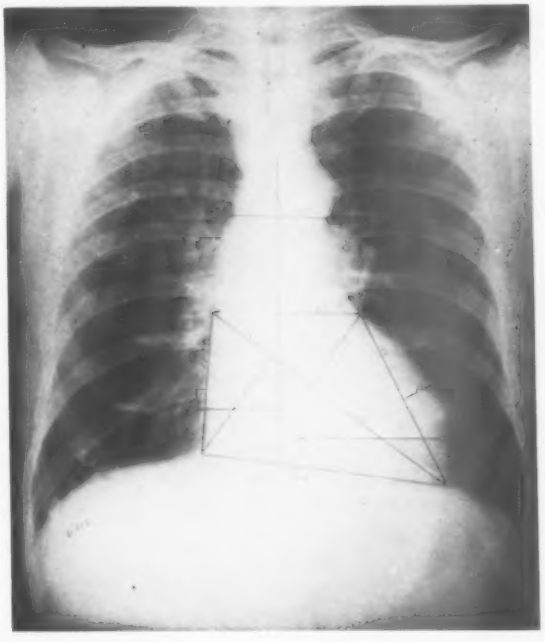


Fig. 7. Aortic stenosis. Hypertrophy and dilatation of the left ventricle, with slow vigorous contractions of the heart and diminished aortic pulsations.

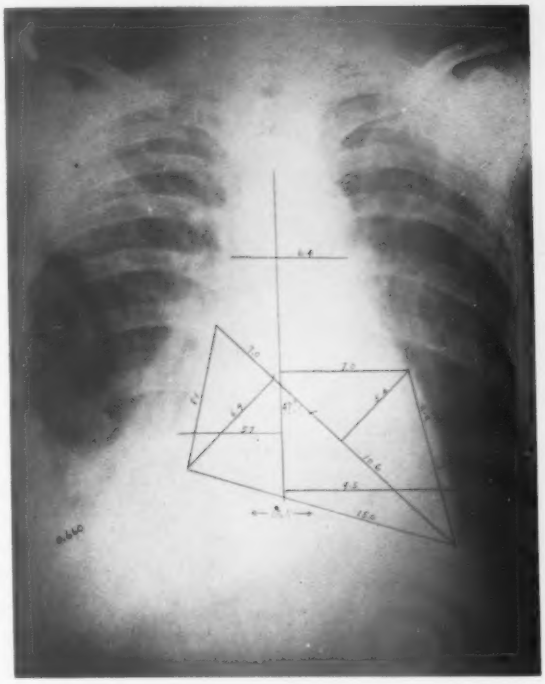


Fig. 8. Aortic insufficiency. Note the increase in the length of the heart due to left ventricular enlargement. The heart shadow is centrally placed and the apex is lowered. The aortic width is increased. Pulsations of the heart and aorta are of increased amplitude.

AORTIC STENOSIS

The changes found in aortic stenosis consist primarily of a narrowing of the aortic orifice. This increases the work of the left ventricle which consequently hypertrophies. The roentgenogram reveals an enlargement of the left ventricle (Fig. 7). The aorta shows practically no change. There is an increase in the left median diameter, the left ventricular measurement and bisector. A normal value for the right

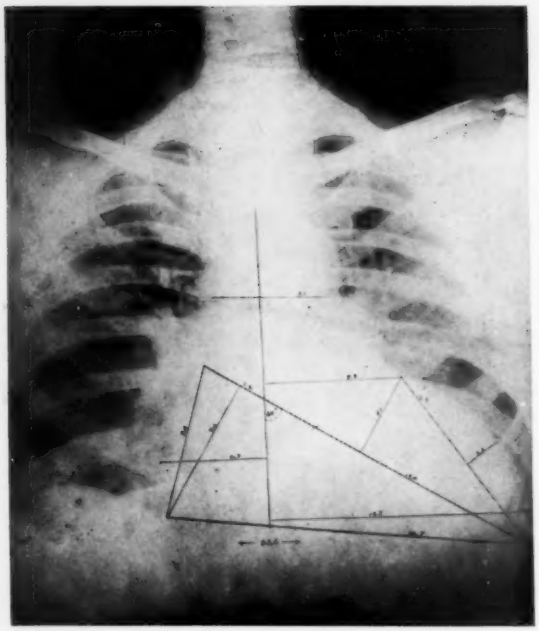


Fig. 9. Tricuspid insufficiency—showing the marked increase in measurement of the right ventricle. Secondary signs are pulmonary and hepatic congestion.

auricular and ventricular cavities will show that these cavities are not enlarged. The length of the heart is increased, and the index of auriculo-ventricular ratio shows this to be due to ventricular enlargement. Roentgenoscopically cardiac pulsations are slow and vigorous, while the amplitude of aortic pulsations is generally diminished.

AORTIC INSUFFICIENCY

Aortic insufficiency allows a reflux of blood from the aorta to the left ventricle. This increases the volume of blood in the left ventricle, which dilates and hypertrophies. The characteristic alteration is therefore left ventricular enlargement. The aortic width is also increased (Fig. 8). Pulsations of the left border of the heart and of the aorta, as seen roentgenoscopically, are of increased amplitude. When dilatation of the left ventricle has progressed to the point where the mitral orifice is stretched, a relative mitral insufficiency ensues. This produces an enlargement of the right heart previously described. Thus a progressive case of aortic insufficiency causes an increase of the entire heart shadow, often of great proportions.

TRICUSPID INSUFFICIENCY

Insufficiency of the tricuspid valve is very uncommon as a single lesion of the heart. It is usually found in association with disease of the left side of the heart or with congenital defects. While the tricuspid valve or its papillary muscles may be involved in an endocarditic process, insufficiency of the valve is more often a "relative insufficiency." It becomes apparent that when the right ventricle is dilated the orifice is stretched so that the flaps no longer suffice to cover it completely. Thus a large value for the right ventricular cavity at once establishes the possibility of a relative insufficiency of the tricuspid valve.

Roentgenographically it is recognized by a considerable increase in the size of the

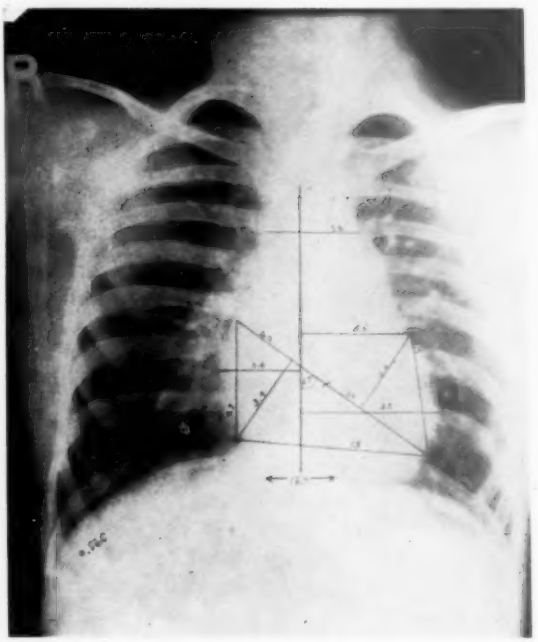


Fig. 10. Pulmonary stenosis. Characterized by increased prominence of the shadow of the pulmonary artery and enlargement of the right side of the heart. Note that the left ventricle is not enlarged, but that the apex is elevated by a hypertrophied right ventricle.

heart, and more particularly, in the size of the right auricle and ventricle (Fig. 9). The measurement of the right auricular and ventricular cavities is increased, the median diameters are increased and the apex is displaced far to the left. The lung fields are more or less congested and the right diaphragm is high, due to hepatic congestion.

PULMONARY STENOSIS

While the picture of congenital pulmonary stenosis is rather characteristic it is difficult to explain its appearance on the basis of mechanical influences, unless, as not infrequently happens, it is associated with persistent ductus arteriosus. There is an exaggerated prominence of the shadow of the pulmonary artery together with an increase in the size of the right chambers. The left ventricle is not enlarged. Its apex is elevated from the diaphragm by the hypertrophied right ventricle (Fig. 10). Roentgenoscopically marked systolic pulsations are observed of the pulmonary shadow and the roots of the lungs. During deep inspiration the right contour of the heart, close to the diaphragm, likewise exhibits systolic contractions which are evidence of an enlarged right ventricle.

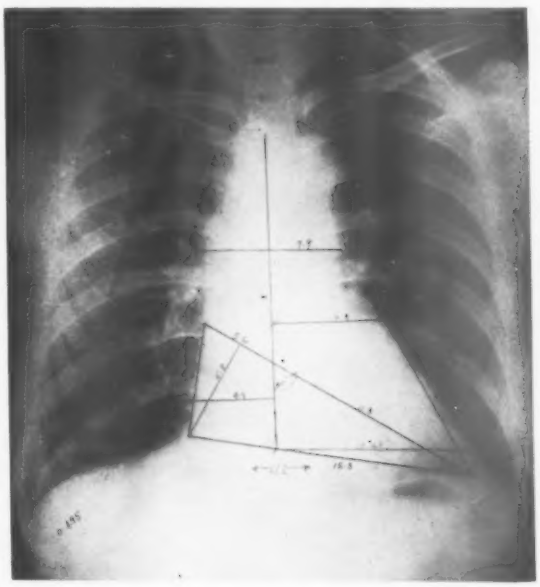


Fig. 11. Aortitis. Increased width of aortic shadow to right and to the left. The "knob" is flattened and broadened. Roentgenoscopically pulsations are of increased amplitude.

Fig. 12. The wide supracardiac shadow simulates the appearance of aortitis (Fig. 11). The heart shadow is that of a mitral incompetence (Fig. 3).

AORTITIS

The appearance of luetic aortitis has previously been described by many writers. We have frequently observed the findings recorded by Holmes,[4] and these have served well to identify the lesion when present. The width of the aortic shadow is increased noticeably to the right. The beginning of the descending portion ("knob") is flattened and broadened (Fig. 11). A perivascular infiltration in the lung immediately adjacent to the aortic shadow is frequently observed. These findings are associated with aortic pulsations of increased amplitude.

It is well to remember that an increase in the diameter of the supracardiac shadow is not necessarily due to the aorta. Figure 12 is of a case of functional mitral insufficiency. This lesion produces no alteration of the aortic contour. Moreover, the changes in the contour of the heart affected by aortic

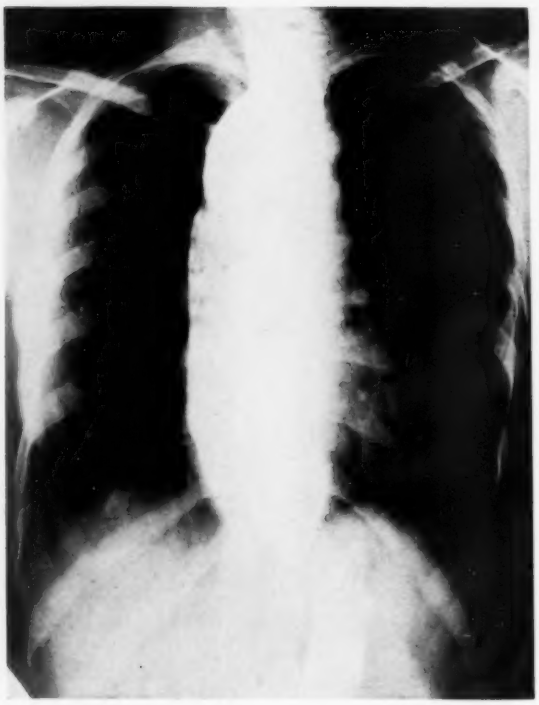


Fig. 13. Same case as Figure 12. Barium meal showing the wide supracardiac shadow to be due to retained food in a greatly dilated esophagus caused by cardiospasm.

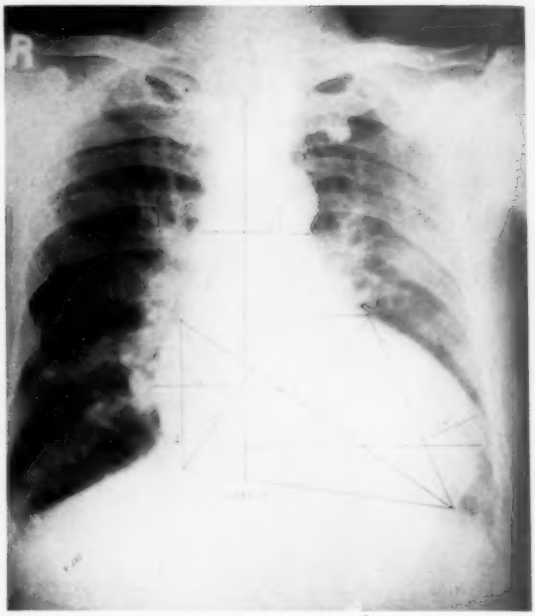


Fig. 15. "Renal heart." In addition to the characteristics of hypertension (Fig. 14) are found considerable hypertrophy and dilatation of the ventricles with evidence of valvular insufficiency.

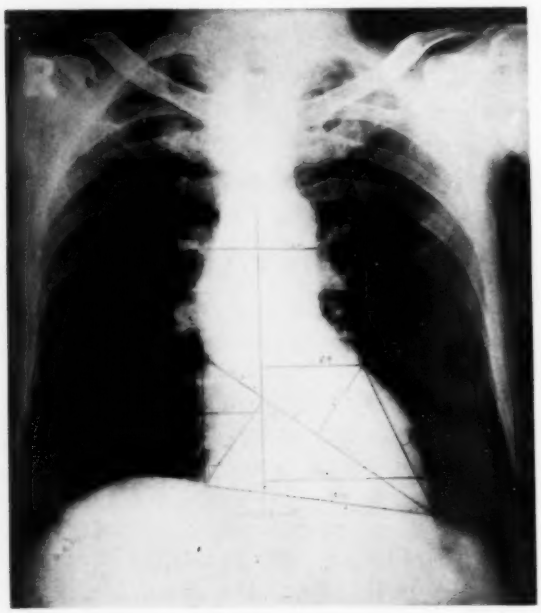


Fig. 14. Hypertension. The junction of the right auricle and vascular arc is sharply accentuated and the curve of the ascending aorta is increased. The left ventricle is frequently enlarged.

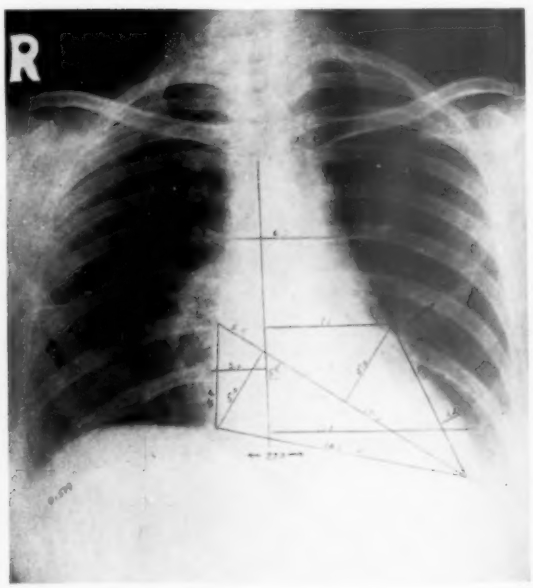


Fig. 16. Thyrotoxicosis. A straightening of the left ventricular contour which becomes concave during deep inspiration (arrow). This appearance is associated with typical roentgenoscopic appearance of *forcible cardiac pulsations*.

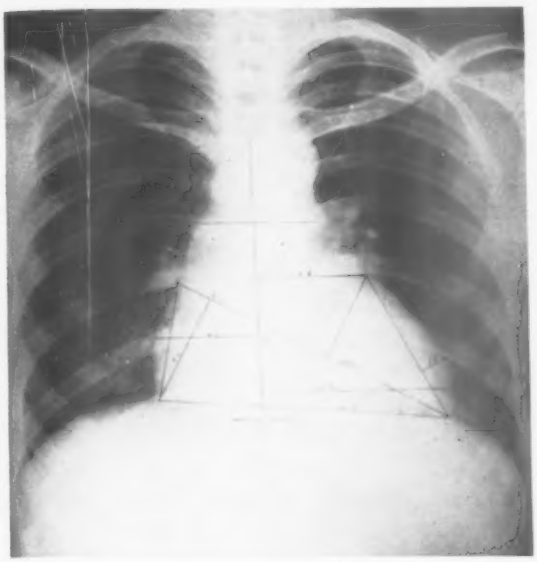


Fig. 17. Myxedema. Symmetrical enlargement of the heart shadow, simulating pericardial effusion (see Fig. 19). The *amplitude of cardiac contractions is approximately normal.*

lesions do not correspond with the appearance presented in this illustration. Further study with the barium meal (Fig. 13) showed the wide supracardiac shadow to be due to retained food in a greatly dilated esophagus caused by cardiospasm.

HYPERTENSION

Cardiovascular hypertension frequently produces changes in the form of the heart which are readily recognized on the roentgenogram (Fig. 14). The ascending aorta appears sharply defined, its right border presenting an accentuation of its usual curve, and the junction between the right auricle and vascular arc is well marked. The left ventricle increases in size. The axis is increased, while the index of auriculo-ventricular ratio is low (0.400–0.650). The ventricular wall is thickened, as is shown by an increase in the value of its bisector (1.3 cm.–2.5 cm.). These changes are observed in so-called "cardio-renal" cases, in which, however, they may be more marked (Fig. 15). Evidence of functional insufficiency of the mitral valve due to progressive increase in the size of the left ventricle is not infrequently associated. The apex is rounded and elevated, partly because of the increased proportions of the left ventricle and partly due to displacement by an enlarged right ventricle. These findings are associated with an increased width and increased pulsations of the aorta.

GRAVES' DISEASE

The manner in which thyrotoxicosis effects changes in the heart is not clearly understood. However, the clinical picture of cardiac embarrassment associated with hyperthyroidism is not uncommon. Vaquez and Bordet have described the roentgenological appearance of the heart in Graves' disease. We have frequently had occasion to confirm their observations.

The characteristic change is found in the left border of the heart (Fig. 16). This consists of a straightening of the cardiac outline, which during deep inspiration becomes concave. The change is best observed at the upper limit of the left ven-

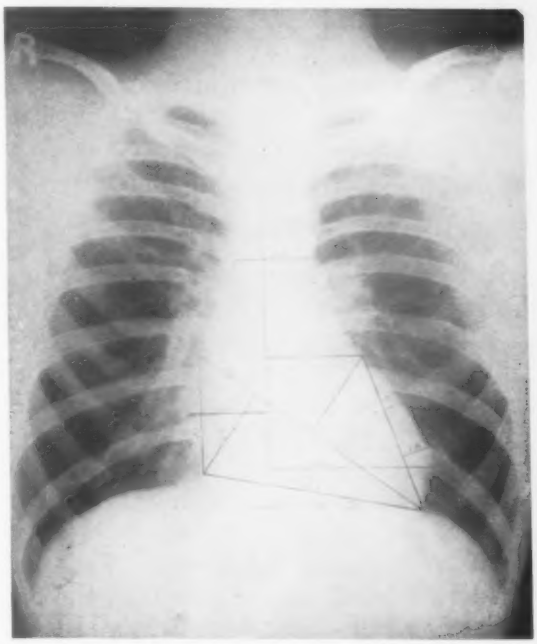


Fig. 18. Same case as Figure 17, taken nine days later, after the administration of thyroid extract. Such rapid involution is typical and confirms the original diagnosis.

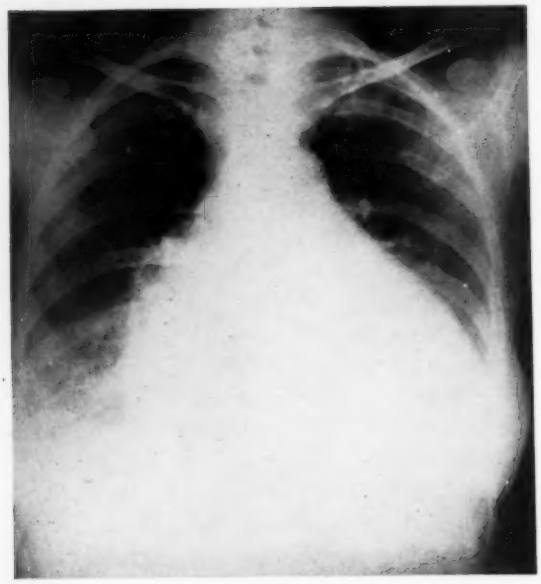


Fig. 19. Pericardial effusion—for comparison with Figure 17. Roentgenoscopically *cardiac pulsations are markedly diminished.*

tricle, near the auriculo-ventricular junction. Roentgenoscopically the heart exhibits contractions of marked amplitude, the rhythm is regular and the rate usually accelerated.

Association with functional disturbances, such as auricular fibrillation or mitral insufficiency, will, of course, alter the characteristic appearance.

MYXEDEMA

Interesting changes in the appearance of the heart are frequently observed in thyroid deficiency.[3,8,12] Roentgenoscopically the amplitude of contraction is but little diminished, the rhythm is regular and the rate approximately normal. The outstanding feature is enlargement of the heart. This is produced by an increase in the value of all its measurements, so that the heart is enlarged symmetrically (Fig. 17). Rapid involution occurs after the administration of an extract of thyroid (Fig. 18). The reduction in size is much more rapid than that observed in other forms of cardiac enlargement.

The general picture is somewhat suggestive of a pericardial effusion. The latter, however (Fig. 19), masks the landmarks of the heart more or less completely, shows a pronounced change in contour with a change in the position of the patient, and exhibits very faint or imperceptible contractions under the roentgenoscope.

ARRHYTHMIAS

Roentgenoscopic examination of the heart includes, in addition to the usual observations made in the anteroposterior and oblique positions, careful study of the rate, rhythm and amplitude of cardiac contractions. We prefer to count the rate by timing with a stop-watch the duration of 20 contractions of the auricle and comparing it with the rate of 20 contractions of the ventricle. This method is not only of value in checking the accuracy of the count but serves to detect irregularities of contraction or conduction which might otherwise be unnoticed.

On the basis of these observations, we have attempted to define the roentgenoscopic appearance of cardiac contractions in the more common conditions. This is graphically illustrated in Figure 20. The

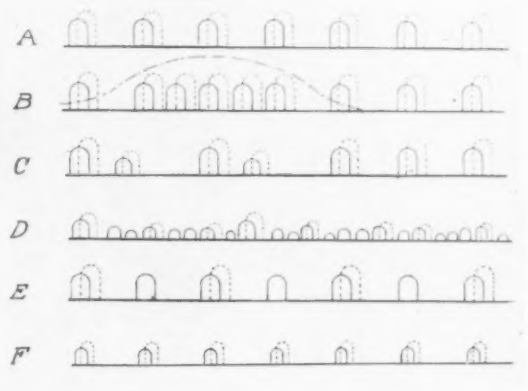


Fig. 20. Roentgenoscopic appearance of cardiac contractions (schematic). Solid curve = auricle; dotted curve = ventricle. Rate is shown by the number, rhythm by the spacing and amplitude by the height of the curves on the base-line. "*A*"—Normal; "*B*"—Sinus arrhythmia (dot and dash curve = inspiration); "*C*"—Extrasystoles; "*D*"—Auricular fibrillation; "*E*"—Heart block; "*F*"—Myocardial impairment.

solid lines represent auricular contractions, the dotted lines being ventricular contractions. Rate is shown by the number of these contractions on the base line, rhythm by their equal or unequal spacing and amplitude by the height of the curves from the base line.

Line "*A*" shows a series of normal cardiac contractions: normal rate, rhythm and amplitude.

Line "*B*" shows the appearance of *sinus arrhythmia*. The long (dot and dash) curve is the duration of one inspiration. It will be seen that during this interval there is an acceleration of cardiac contractions. The latter, however, retain uniform rhythm and normal amplitude, i.e., while the rate may change momentarily from 80 per minute to 100 per minute, and then back again to 80, contractions are rhythmic and their force is unchanged. These hearts show no abnormality in contour (Fig. 21).

Line "*C*" presents the appearance of *extrasystoles*. During a series of normal contractions is observed an extrasystole. This comes very closely upon the completion of a normal contraction and is followed by a pause. Thus the dominant rhythm is momentarily disturbed but reverts immediately to its normal phase. If the heart rate is accelerated (as by exercise) extrasystoles tend to disappear. Roentgenographically these hearts present no alteration in contour (Fig. 22).

Fig. 21. Sinus arrhythmia. There is no alteration of cardiac contour or size.

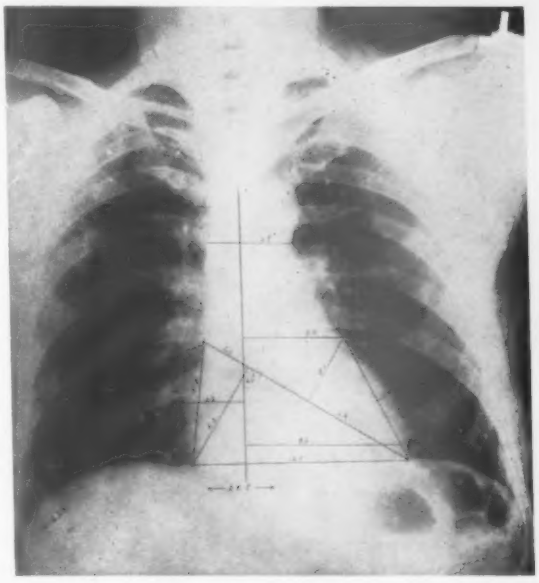


Fig. 22. Extrasystoles. There is no alteration of cardiac contour or size.

Line "*D*" is a schematic representation of the roentgenoscopic appearance of *auricular fibrillation*. The general amplitude of contractions, particularly of the auricles, is greatly diminished. This may account for the claim by some observers* that no contractions are visible in this condition. The rhythm is irregular and the rate fluctuates. Contractions frequently appear as faint undulations. These irregularities are independent of respiratory influence. It is usual to obtain a rate of 140 a minute or more. While such a rate does not correspond with that obtained by electrocardiography, it appears that not all the impulses which are of sufficient intensity to produce measurable electrical impulses are of sufficient magnitude to in-

* Holmes and Ruggles. Roentgen Interpretation. Lea and Febiger, Philadelphia, 1926.

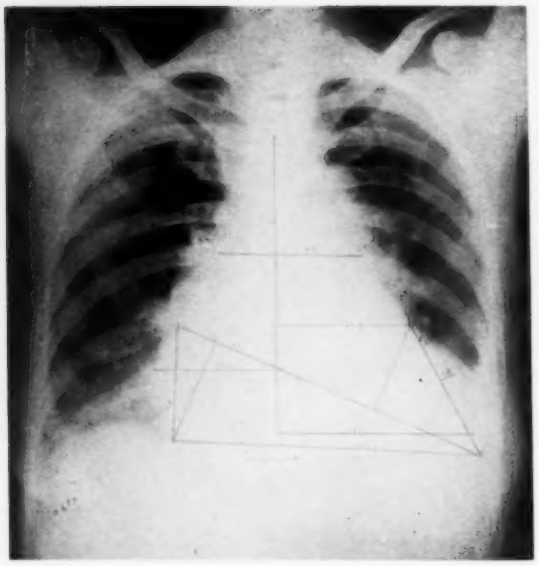


Fig. 23. Auricular fibrillation. The area of the auricles is increased.

duce physical contraction of the auricles. Moreover, the impulses which induce auricular contraction are not all followed by ventricular contraction, either because of

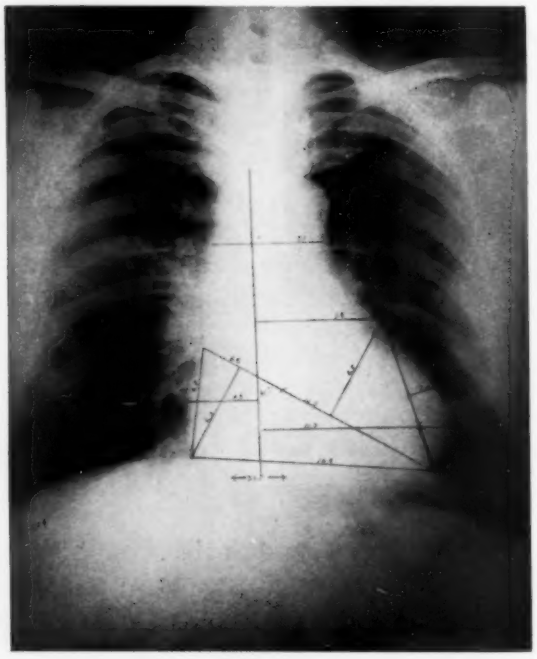


Fig. 24. Heart block. Hypertrophy and dilatation of the left ventricle appear as a compensating mechanism.

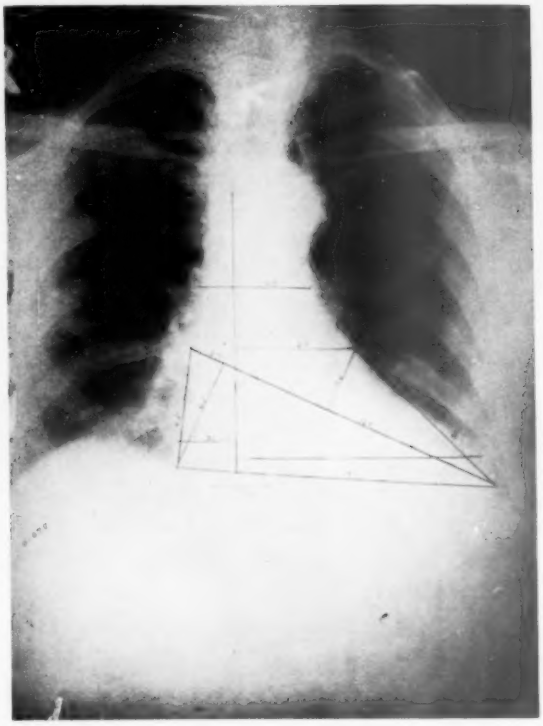


Fig. 25. Myocardial impairment. The median diameters are close to the diaphragm, the left border of the ventricle is straight or concave and the bisector is small. Roentgenoscopically cardiac contractions are greatly diminished.

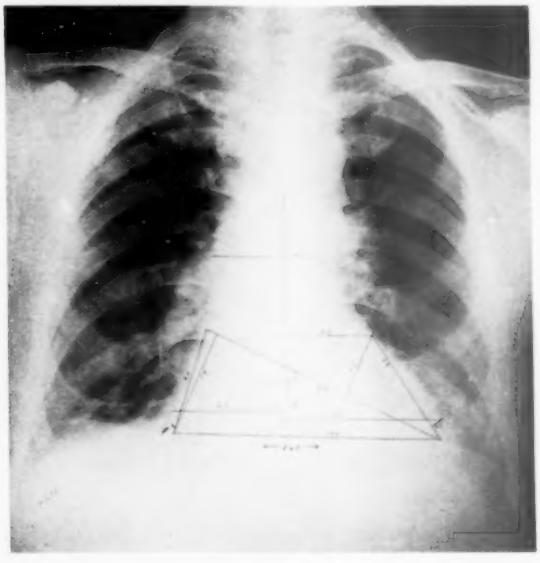


Fig. 26. Coronary thrombosis, showing the typical picture of myocardial impairment (see Fig. 25), the cause of which is derived by exclusion of toxemia, endocarditis and poisoning, as etiologic factors.

the inadequacy of the stimulus or because of impaired auriculo-ventricular conduction. Arrhythmia is always present unless complete heart block exists (such as may be induced with digitalis), in which case ventricular contractions may be regular but slow. The roentgenogram (Fig. 24) shows enlargement of the auricles.

Line "*E*" shows the appearance of typical *auriculo-ventricular block*. In the illustration 20 auricular systoles were timed in a period (fifteen seconds) exactly one-half the duration of 20 ventricular systoles (thirty seconds); thus a ratio of two to one. This slow ventricular rate (40 per minute) is characteristic of complete heart block.

If this type of arrhythmia persists, a compensating mechanism in the form of hypertrophy, particularly of the left ventricle, develops. This change is identified on the roentgenogram (Fig. 23) by an increase in the left median diameter, the size of the left ventricular cavity and its bisector. The index of auriculo-ventricular ratio is low.

The diagnostic problem presented by cardiac enlargement without such alterations of contour as are usually associated with valvular lesions is therefore clarified on the basis of two findings: a rate of auricular contraction which may be normal or increased, with a rate of ventricular contraction which is diminished, and left ventricular enlargement—both identified with heart block.

Line "*F*" shows the type of contraction seen in *myocardial impairment*. The amplitude of contractions is markedly diminished: the greater the myocardial damage, the weaker the contractions. The roentgenogram (Fig. 25) shows typical changes. Instead of the usual convex left border of the heart, the latter is straight or concave. The bisector is very much reduced. The left and right median diameters appear almost in the same straight line and lie close to the diaphragm. The heart appears to "sag."

A marked diminution in the amplitude of contractions is observed in coronary thrombosis. The general picture seems fairly typical. However, the not infrequent association of myocardial damage with endocarditis or toxemia makes it difficult to decide, in those cases which present appreciable alteration of cardiac contour, whether coronary thrombosis is present or not.

The presence of coronary thrombosis unassociated with valvular lesions or toxic manifestations is characterized by the appearance of feeble contractions and a cardiac outline which cannot be identified with the deformations of valvular lesions. The roentgenogram (Fig. 26) usually shows the presence of myocardial impairment, although occasionally the heart outline may show no recognizable departure from normal.

When coronary thrombosis constitutes only one element in a case of heart disease, its identity cannot be specifically established; rather, the more categorical entity of "myocardial damage" appears to be the limit of the present diagnostic abilities of the roentgen ray in such cases.

COMMENT

All of the diagnoses in this work were made from roentgenoscopic examination and studies of the roentgenograms alone.* It is apparent that when combined with complete clinical data the roentgen ray is an invaluable aid in cardiac diagnosis.

We desire to point out some of the difficulties encountered, such as may introduce potential sources of error in interpretation.

Not infrequently the rebound from ventricular systole is of sufficient magnitude to cause movement, simulating contraction of the auricles. If, by error, this movement is counted as an auricular contraction, the auricular rate will be a multiple of the ventricular rate (e.g., 2 to 1). The rate, however, will be too rapid for heart block and too slow for paroxysmal tachycardia or flutter. The rhythm will be

* It was necessary to use clinical data to establish a diagnosis of the case represented by Figures 17 and 18.

too regular for auricular fibrillation, and likewise too regular for extrasystoles or for ventricular dropped beats. The error therefore can be readily recognized.

When the amplitude of contraction is so slight as to make a count difficult, it is frequently possible to obtain better roentgenoscopic visualization by studying contractions in the oblique view most suitable for presentation of the desired portion of the heart.

Exact localization of the points on the cardiac outline needed for measurement is essential. It is apparent, for example, that if the apex is placed too high, the length of the heart will be reduced, and this will be shown as a decrease in the size of the ventricles, so that a high index of auriculoventricular ratio, indicating auricular enlargement, is erroneously obtained. Roentgenoscopic examination is therefore essential not only in studying the activity of the heart, but in the localization of the landmarks needed for measurement. A tracing made at the time of this examination serves to locate these points on the film.

A guide to the differentiation of the causative factors in a given case of cardiac enlargement is frequently found in the simple formula that the lesion is usually distal to the enlargement. Thus enlargement of the right side of the heart is most frequently due to mitral lesions, defects of the interventricular septum and pulmonary stenosis. The left auricle becomes enlarged with lesions of the mitral valve. Enlargement of the left ventricle is due to aortic disease, nephritis, chronic hypertension, lead poisoning, and, what appear to be exceptions to the "rule," heart block and mitral insufficiency.

CONCLUSIONS

The value of the roentgen ray in the diagnosis of heart disease does not end with the detection of cardiac enlargement. With increasing experience it is possible to derive from careful roentgenographic and roentgenoscopic studies specific information regarding the presence and identification of valvular disease, the integrity of the myocardium and the functional state of the heart as a whole.

Distinctive cardiopathies usually produce characteristic alterations in the form of the heart. These changes may be shown by measurement of the heart shadow and of its component parts, revealing not only a gross enlargement of the organ, but an altered interrelationship of its chambers, making it frequently possible to identify the anatomical lesions of the heart.

Roentgenoscopic examination permits of an appraisal of the functional integrity of the heart and the identification of the various arrhythmias.

It is apparent that the interpretation of these changes depends not only upon careful roentgenologic technique, but equally upon a working knowledge of the clinical and pathological aspects of heart disease.

REFERENCES

1. Assmann, H. Die klinische Röntgendiagnostik der inneren Erkrankungen. F. C. W. Vogel, Leipzig, 1924.
2. Dietlen, H. Herz und Gefässe im Röntgenbild. J. A. Barth, Leipzig, 1923.
3. Hertoghe, E. Thyroid deficiency. *Med. Rec.*, 1914, *86*, 489–505.
4. Holmes, G. W. The use of the x-ray in the examination of the heart and aorta. *Boston M. & S. J.*, 1918, *179*, 478.
5. Kaufmann, E. Pathology for Students and Practitioners. Translated by S. P. Reimann. Blakiston's Son & Co., Philadelphia, 1929.
6. Köhler, Alban. Roentgenology. Wm. Wood & Co., New York, 1929.
7. Mackenzie, J. Diseases of the Heart. Oxford Publications, Freude, 1925.
8. Reid, W. D., and Kenway, F. L. Electrocardiographic signs associated with low basal metabolism. *Endocrinology*, 1929, *13*, 191–204.
9. Rummo. Sulla cardio topometria. *Reforma med.*, 1898, *2*, 533.
10. Vaquez, H., and Bordet, E. The Heart and the Aorta. Translated by James A. Honeij and J. Macy. Yale University Press, New Haven, 1920.
11. Vaquez, Henri. Diseases of the Heart. Translated by G. F. Laidlaw. W. B. Saunders Co., Philadelphia, 1924.
12. Zondek, H. Das Myxödemherz. *München. med. Wchnschr.*, 1918, *65*, 1180–1182.

Vol. XXVIII, No. 4

CONGENITAL ARTERIOVENOUS COMMUNICATION*

By F. G. LINDEMULDER, M.D.
Instructor in Neurology, University of Michigan Medical School
ANN ARBOR, MICHIGAN

CONGENITAL arteriovenous communication is a rare anomaly and may be situated in various parts of the vascular system. Callander, in his comprehensive study of 447 cases of arteriovenous fistula, found only 3 to be of congenital origin. Lewis, ten years later, in 1930, reviewed the literature and found only 24, to which he added 5 cases which he considered to be congenital arteriovenous fistula. In 1932, Horton collected 24 additional cases in which he emphasized the presence of hypertrophy of the extremities. In the discussion of this paper, Lewis stated that he believed hypertrophy of bone and soft tissues was common in the affected extremity. Dandy, in his work on arteriovenous aneurysm of the brain, demonstrated ventriculograms showing dislocation of the ventricular system away from the lesion.

Brown, Yater, Pemberton and Saint, and others, believe the O_2 content of the blood in the regional veins shows an increase and have used this as an aid in the diagnosis of arteriovenous communications. The bradycardiac reaction, first described by Branham and later by Nicolandi, consists of slowing of the heart rate when a large artery in the involved extremity is occluded.

Two cases are here reported, the first is of special interest because of the deviation of the ventricular system toward the side of the lesion, and the second case because it is one in which there is a hypertrophied extremity.

CASE 1. R. C., male, aged thirteen, was admitted to the Neurological Service of the University Hospital in April, 1930, with a history of epilepsy. The attacks seemed to definitely affect the right more than the left side of the body. His seizures had been present since the age of five years. The past and family histories were irrelevant. He was cooperative, but a little

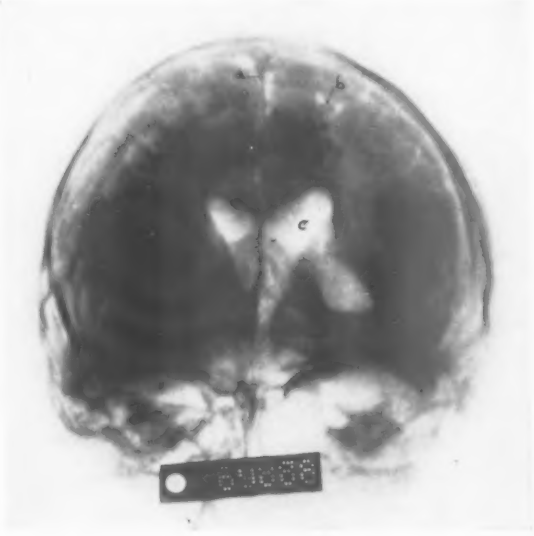


FIG. 1. Case 1. Anteroposterior view: (*a*) falx cerebri deviated to the left; (*b*) cortical atrophy; (*c*) dilated left ventricle.

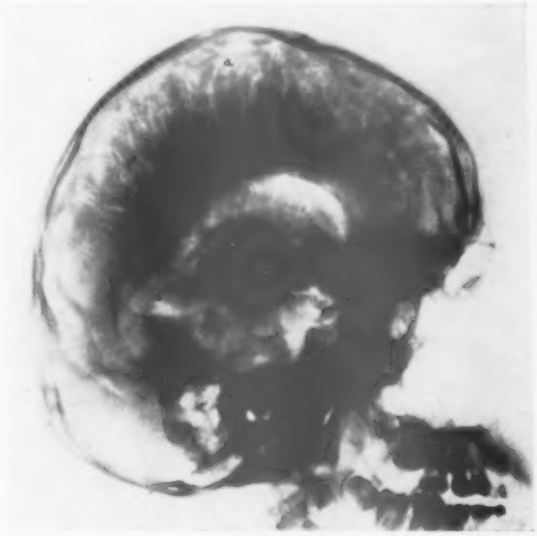


FIG. 2. Case 1. Lateral view: (*a*) marked cortical atrophy; (*b*) posterior horn of dilated left lateral ventricle.

*From the Neurological Service of the University Hospital, Ann Arbor, Michigan

impatient and somewhat distractible. The intelligence quotient was 69. The neurological and general physical examination revealed nothing. The eye grounds showed a slight paling of the discs. The spinal fluid and blood Kahn reactions were negative.

An encephalogram with the patient in the sitting position revealed a cerebrospinal fluid pressure of 450 millimeters of water; 120 c.c. of cerebrospinal fluid was withdrawn and 135 c.c. of air injected. Encephalographic studies showed, in the anteroposterior view (Fig. 1), the left lateral ventricle to be dilated and slightly deviated from the midline. The falx cerebri was dislocated toward the left. There was more evidence of cortical atrophy on the left than on the right, as shown by the presence of more air in the subarachnoid spaces. In the lateral projection (Fig. 2) the marked dilatation of the left lateral ventricle was seen, while the right appeared to be about normal in size. There was a definite cortical atrophy, particularly on the left side.

The patient was operated upon by Dr. Max M. Peet, who turned a left osteoplastic flap exposing the parietal region. A cluster of blood vessels was exposed over the left cerebral cortex. Here the small arteries over the surface of the brain were considerably enlarged. The veins were likewise enlarged, and by milking the blood out of them, it was seen that they immediately filled with arterial blood.

Comment. In this case the encephalographic findings demonstrated dilatation of the left lateral ventricle and cortical atrophy, with deviation of the falx cerebri toward the side of the lesion. These findings are the opposite of those described by Dandy in ventriculography. An explanation for the changes may be due to the presence of cortical atrophy which resulted from a previous hemorrhage on the left side of the brain.

Case II. K. K., female, aged thirty-four, was first examined at the Neurological Service of the University Hospital on October 28, 1931.

Fig. 3. Case II. Variation in the size of bones and supporting tissues.

Her condition was diagnosed as a psychoneurosis. During the examination it was noted that she had a large arm and was sensitive when this was mentioned. However, there were no other complaints referable to the extremity. The enlarged right upper extremity was noted by the mother shortly after birth. She was able to trace her family history for three generations, and there was no evidence of a similar condition or of any nervous or mental disease. Our patient was the fifth child and it was said that the mother had a normal delivery at her birth, and no instruments were used. The patient has one child aged eleven, who has no anomalies or deformities other than a scar on the head which was caused from forceps at delivery.

The neurological and general physical examination was entirely negative except for the right upper extremity. The anthropometric measurements are as follows: the upper extremities, from the acromion process to the tip of the middle finger, measures: right, 29.75 inches; left, 28.5 inches. Girth of the right hand, 8.5 inches; left, 7.25 inches. Girth of the right forearm, 10 3/8 inches; left, 9 inches. Girth of the right arm, 11 inches; left, 10 3/8 inches. The surface veins on the anterior aspect of the right arm were prominent and large. The oxygen content of the venous blood of the right arm was 15.8 volumes per cent; left, 12.9 volumes per cent. No thrills or bruits were heard. The bradycardiac reaction showed a decrease in the heart rate of 4 per minute when the brachial artery was occluded. There was only one marked variation of the skin temperature and that was on the dorsum of the right hand which was 29.8°C., while the left was 28.6°C. Roentgen studies of both hands on the same film showed the variation in size of the two hands (Fig. 3).

Comment. Congenital hemihypertrophy of an extremity is exceedingly rare. Gesell in a careful review of the literature in 1921 and again in 1927 found 53 cases. This case is of special interest because it is probable that the asymmetry was caused by arteriovenous communications.

SUMMARY

Two cases are reported of congenital arteriovenous communication. The first demonstrates that in encephalography the enlarged lateral ventricle is on the side of the lesion and the deviation of the brain is toward the side of the arteriovenous communication. This finding is opposite to that previously described in ventriculography. The second is one in which there is a hypertrophy of an extremity, this variation being demonstrated in the roentgen studies of the two hands.

REFERENCES

1. BROWN, G. E. Abnormal arteriovenous communications; diagnosed from oxygen content of the blood of regional veins. *Arch. Surg.*, 1929, *18*, 807–810.
2. CALLANDER, C. L. Study of arteriovenous fistula with an analysis of 447 cases. *Johns Hopkins Hosp. Rep.*, 1920, *19*, 259–358; also *Ann. Surg.*, 1920, *71*, 428.
3. DANDY, W. E. Arteriovenous aneurysm of the brain. *Arch. Surg.*, 1928, *17*, 190–243.
4. GESELL, A. Hemihypertrophy and mental defect. *Arch. Neurol. & Psychiat.*, 1921, *6*, 400–425.
5. GESELL, A. Hemihypertrophy and twinning. *Am. J. M. Sc.*, 1927, *173*, 542–555.
6. HORTON, B. T. Hemihypertrophy of extremities associated with congenital arteriovenous fistula. *J. Am. M. Ass.*, 1932, *98*, 373–379.
7. LEWIS, D. Congenital arteriovenous fistula. *Lancet*, 1930, *2*, 621; 680.
8. NICOLANDI. Phlebarteriektasie der oberen Extremitat. *Arch. f. klin. Chir.*, 1875, *18*, 252.
9. PEMBERTON, J. DE J., and SAINT, J. H. Congenital arteriovenous communications. *Surg., Gynec. & Obst.*, 1928, *46*, 470–483.
10. YATER, W. M. Acquired arteriovenous fistula. *Ann. Surg.*, 1928, *87*, 19–31.

A STUDY OF THE HIP JOINT FROM THE STANDPOINT OF THE ROENTGENOLOGIST*

By L. B. MORRISON, M.D.
BOSTON, MASSACHUSETTS

THE hip joint has been the object of study for physicians from earliest times, principally because of its economic value, when normal, in maintaining man at his best for competing in the struggle for existence. In the Fourth Century, B.C., the hip joint was quite extensively described by the Hippocratic School. Later, in the Second Century, A.D., Galen of Pergamum added a description of the compact bony elements. Filippo Ingrassias described the epiphyseal margins of the innominate bone about the middle of the Sixteenth Century, A.D. Even such fine anatomical points as the os acetabulum were known and described by Bernard Albinus in the Eighteenth Century. Interest today is far from lacking, as is evidenced by plentiful articles found in the literature. These have by no means exhausted the field, and there yet remain many lengthy and complex problems. Only through the pooling of individual knowledge, and by careful sorting and testing of these ideas can we hope to reach correct conclusions. Today, I wish to set before you some observations I have made on the subject. I hope they may be of use in clearing up a few of the complexities confronting us. The picture I shall lay before you is of necessity seen through the eyes of a roentgenologist.

Included in this study are the acetabulum and the anterior inferior spine of the ilium, the articular head of the femur, the neck, the major and minor trochanters, because certain lesions arising in relation to these areas are significant.

The coxal or hip joint is an enarthrodial (ball and socket) joint, formed by the reception of the head of the femur into the cup-shaped cavity of the acetabulum.

The innominate bone is composed of the ilium, ischium and pubis, and part of each is required to form the acetabulum. The latter is a deep cup-shaped, hemispherical depression, directed downward, laterally and forward. Its mesial one-fifth is pubis, superior two-fifths, ilium and its lateral and inferior two-fifths, ischium. The acetabulum is bounded by a prominent, uneven rim which attaches the glenoid labrum. Inferiorly, in the rim is the acetabular fossa, and through this notch nutrient vessels and nerves gain the ligamentum teres leading to the head of the femur. The remaining acetabulum forms the lunate surface which is the bearing surface for the ball-shaped head of the femur. Anterior and superior to the acetabulum is the anterior inferior iliac spine which rises above the iliopectineal eminence to originate the straight head of the rectus femoris muscle.

Anatomists tell us that there are three primary centers and two secondary centers concerned in the ossification of the acetabular region. The three primary centers consist of one for each of the three main bones. One secondary center is concerned with the ossification of the anterior inferior iliac spine. The other secondary center, with which we are concerned, is found in the Y-shaped piece of cartilage at the junction of the three main bones in the bottom of the acetabulum. This may be divided into a center for the os acetabuli, which is the portion between the ilium and pubis, and one or more for the remaining Y-shaped cartilage. This Y-shaped cartilage allows for the expansion of the acetabulum. At birth, the three primary centers are separate.

About the thirteenth or fourteenth year, the three primary centers have extended their growth into the bottom of the acetabulum and are there separated from each other by a Y-shaped portion of cartilage,

* Read at the Thirty-second Annual Meeting, American Roentgen Ray Society, Atlantic City, N. J., Sept. 22-25, 1931.

which now presents traces of ossification, often by two or more centers. One of these, the "os acetabuli," appears about the age of twelve, between the ilium and pubis, and fuses with them about the age of eighteen; it forms the pubic part of the acetabulum. The ilium and ischium then become joined, and lastly the pubis and ischium, through the intervention of this Y-shaped portion. At about the age of puberty, ossification occurs in each of the remaining portions, and they join with the rest of the bone between the twentieth and twenty-fifth years.

We wish to demonstrate the existence of an additional genetic center on the superior acetabular rim. This may be divided into an anterior, a superior, and a third posterior segment. Its function is to widen the upper semicircular portion of the rim and thereby deepen the acetabular cavity for the reception and retention of the head of the femur. This center appears constantly in a great majority of plates of normal hips of children between the ages of ten and seventeen, and is found joined to the completed rim by the age of eighteen or, at times, earlier.

These accessory acetabular epiphyses were reported by me on December 13, 1926, before the New England Orthopedic Society. Since then, I have endeavored to find some substantiation in works of anatomists for these bony elements. These epiphyses have escaped the anatomist principally because subjects between the ages of ten to seventeen are not often available. If they have been found in dissections, they were undoubtedly considered anomalous. Gray, Cunningham, Piersol, Sobotta and other available anatomical texts fail to describe any such elements.

The possibility of these epiphyses has not been entirely overlooked. In 1864, Rambaud and Renault published "Development des Os" with an accompanying atlas of plates. One of these plates, showing an acetabulum, hints at this secondary rim construction. The descriptive text speaks of "an invasion of bony elements into the cartilage of the acetabular margin which set up secondary centers of bony material around the acetabular rim." They are described as "forming where the muscular and ligamentous insertions are most numerous." According to these authors, these bony invasions "first appear about twelve years of age and fuse at the age of fifteen."

Lilienthal, in 1909, reported this accessory nucleus. He, however, was of the opinion that it was a bone nodulus.

Fromme shows a roentgenogram of this accessory bone center, calling it the "x-ray os acetabuli," to distinguish it from the anatomical os acetabuli. He agrees with Brand that "it arises as a result of fracture or a reconstruction of bone—due to bone softening or excessive stress." "They are supposed in a majority of cases to be observed as a consequence of secondary rickets or coxa vara adolescentium."

Meyer, in "Röntgendiagnostik in der Chirurgie und ihren Grenzgebieten," published in 1927, writes:

> There is mentioned quite frequently in radiological literature an OS ACETABULI which lies above the acetabular margin and which can scarcely be identical with the os acetabuli. There is an accessory nucleus on the acetabular margin accepted by different authorities (Nieber, Stieda, Lilienthal, Grashey), which probably is identical with that os cotyloideum superior described by Perna on the upper acetabular wall. According to Perna, this nucleus is visible between the ages of nine to twelve years and disappears by the ages of seventeen to twenty-one. Concerning the frequency and form of the os cotyloideum superior, up to now nothing is known roentgenologically. Its production seems dependent upon the extension of the projecting surface. An os acetabulum can exist without secondary rickets or bone softening. The decision whether there are centers of ossification, or bone reconstruction, is not possible to be determined alone by x-ray pictures. Most apparent appears the existence of a normal ossification center in the acetabular rim of youths between the ages of ten and seventeen years.

These references demonstrate that much doubt exists concerning the significance of these bony structures. Studies of series of

Fig. 1. Normal hips at birth.

plates have led us to conclude that these accessory epiphyses are important in determining the final structure of the adult acetabulum.

Therefore, we conclude that the acetabulum develops and expands through three agencies. First, by the bony centers found in the os acetabulum and the remaining Y-shaped portions. Secondly, by the epiphyses of the ilium, ischium and pubis whose junction determines this cavity. Thirdly, by the existence of the three separate bony centers arising on the anterior, superior and posterior portions of the acetabular rim. This last agency of growth determines the final deepening of the normal acetabulum. Its failure to develop normally in any part, or its overgrowth, explains the shallow acetabulum, or the exaggeratedly deep acetabulum, respectively.

The head of the femur, which is contained normally in the acetabulum, is slightly more than hemispherical and is covered with cartilage except for its central fovea. It is noteworthy that the bony structure of this head is as dense on the inferior portion of the ball as it is on the superior portion. This demonstrates that the pressure is divided equally over the bearing surfaces of the sphere resting on the lunate portion of the acetabulum.

The neck of the femur is continuous with the head superiorly and the shaft inferiorly at an obtuse angle. This union of the neck and shaft is marked by a greater and lesser trochanter.

The ossification centers of these portions of the femur are four: one for the head, one for the body (including the neck), and one for each trochanter. The epiphyses unite after puberty, the lesser trochanter first, then the greater, and finally the head, about the eighteenth year.

Frequently we observe an additional bone center located on the tip of the great trochanter. It is not present in all cases and varies greatly in extent of development. We believe that here is found the explanation for the wide variation found in size and shape of individual great trochanters. We have never observed a similar bony center on the lesser trochanter.

Let us now view the development of the normal hip as interpreted by the roentgenologist. For this discussion I have chosen what I consider the average normal of a series of roentgenograms taken of hips of all ages.

The acetabulum begins to be formed by three cartilaginous centers about the seventh week. These three cartilaginous elements,

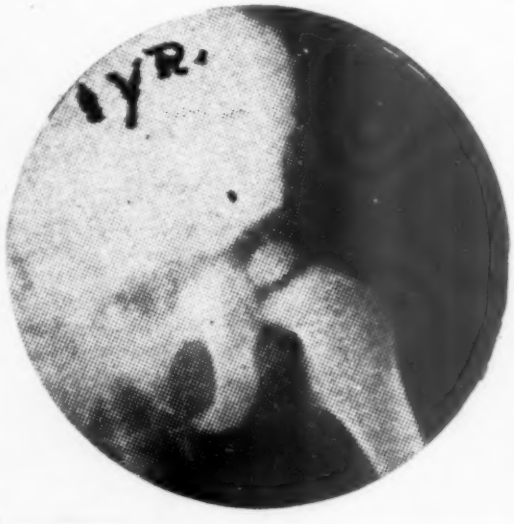


Fig. 2. Normal hip at one year of age.

ilium, ischium and pubis, meet in a Y-shaped acetabular suture. Towards the end of the second month, cartilaginous outgrowths join these three elements to form the shallow acetabular cup. At this stage, the roentgen ray shows little, but it must be remembered that the hip joint is present and is completely laid down in cartilage which is radiolucent.

At birth (Fig. 1), the acetabulum appears as a wide V-shaped notch with bony framework starting with the anterior inferior iliac spine, and extending downward and around to the wide open cartilaginous space at the junction of the ilium, ischium and pubis. The inferior portion slopes downward and outward to the most inferior tip of the ischium. Around these very scanty landmarks the acetabulum and head of the femur must be pictured as laid down in cartilage. At this time, there is no capital epiphysis seen on the femur, the lesser trochanter is absent, and only a rudimentary great trochanter appears.

At one year of age (Fig. 2), the acetabulum is deepened and the Y-shaped cartilaginous space at the junction of the ilium, ischium and pubis is narrowed. The capital epiphysis of the femur is well formed, and definite lines of demarcation occur for both major and minor trochanters. As yet these latter show no ossifying centers. The acetabular cavity, superiorly, extends to the anterior inferior iliac spine, since no ossification has occurred in the ilium below this point. More bone appears at the expense of the preformed cartilage existing in the originally shaped acetabulum.

Fig. 3. Normal hips at two years, two and one-half months of age.

Fig. 4. Normal hips at three years of age.

At two years and two months (Fig. 3), the configuration of the acetabulum is well started and is almost circular. The superior or bearing portion, belonging to the ilium, is outlined with dense margins. The Y-shaped cartilage supporting the ilium, ischium and pubis is distinct, but much lessened. There is a regular acetabular rim composed of bony and considerable cartilaginous elements. The capital epiphysis of the femur is larger, the conjugal cartilage is very wide, but as yet no epiphysis has been formed for the great trochanter.

At three years of age (Fig. 4), there is a definite dense rim around the margin of the acetabulum. The bony portions of the acetabulum appear shallow, because the anterior and posterior margins are still cartilaginous. The superior or bearing portion of the acetabulum is shelf-like and the acetabular socket appears completely formed, superiorly. This, however, will be shown to represent a stage at which the ilium has developed laterally to its greatest extent with its present ossification center, whereas the femoral head has only begun its process of growth. A decided space exists between the articular surface of the

Fig. 5. Normal hips at four years of age.

capital epiphysis of the femur and the bony margin of the superior acetabulum which is formed principally of the ilium. The Y-shaped cartilaginous junction of the ilium, ischium and pubis is still distinct and has no ossification center. The great trochanter is first noted as having an ossification center.

At four years of age (Fig. 5), there is very little change in the acetabulum from that of three years, except for continued ossification of the cartilaginous portions and a narrowing of the Y-shaped cartilage area of the posterior and anterior portions of the acetabulum between the ilium, ischium and pubis. The great trochanter has shown decided growth and the capital epiphysis

Fig. 6. Normal hips at five years of age.

is well formed and rounded. At this stage, the superior margin of the acetabulum is rounded and smooth, showing diminished ossifying activity.

At five years (Fig. 6), the acetabular rim and margins of the cartilages show progressive ossification. Since new bone forms from the ever branching periosteal buds, the Y-shaped cartilage dividing the ilium, ischium and pubis looks more ragged than before. The capital epiphysis is becoming larger. Its conjugal cartilage is more ragged appearing in outline. The prominence of the lesser trochanter is becoming more marked, and the anlage of the anterior inferior iliac spine can be observed.

Fig. 7. Normal hips at six years of age.

At six years of age (Fig. 7), the shape of the acetabulum appears rounded, the outer margin is fairly smooth and the Y-shaped cartilage at the junction of the ilium, ischium and pubis is narrowed; but the anterior and posterior acetabular rim cannot be made out. These remain cartilaginous bearing surfaces. The epiphysis in the upper end of the femur is distinct and wide. The epiphysis of the greater trochanter is well developed and smooth. The diaphyseal portion of the lesser trochanter can be observed to bulge to anticipate the epiphyseal bud.

About the seventh year (Fig. 8), the ossifying centers in the acetabulum send their periosteal buds out into the cartilage

in limited numbers and the rim regains its smooth appearance. Only a slight advance in growth of the epiphysis at the head of the femur and those of the major and minor trochanters is observed. At this age, the density of the bone in the acetabular rim has increased noticeably. It is of interest that that part of the rim belonging to the ilium has become most dense.

At eight years (Fig. 9), the acetabular margin is little changed. There is continued fusing together of the ossification centers of the cartilage, and the wide V-shaped notch originally observed as the primitive acetabulum is now replaced by bone which grows from above downward. The epiphyseal lines are consequently narrowed. The epiphysis of the lesser trochanter is observed with a distinct cartilage line between it and the diaphyseal side. The great trochanter retains its smooth shape and its epiphysis is distinct. The conjugal cartilage line of the head of the femur is clear.

Fig. 9. Normal hips at eight years of age.

Fig. 8. Normal hips at seven years of age.

At the ninth year (Fig. 10), the whole acetabulum becomes relatively deepened and there is little cartilage left in the Y-shaped junction of the ilium, ischium and pubis. The acetabulum retains the smooth appearance, denoting lessened activity in bone growth.

Again at the tenth year (Fig. 11), the increased trabeculation of the acetabular cavity is observed. This appearance seems due to new bony invasions occurring to speed the ossification of the remaining cartilage. The surface margin of the acetabulum looks more radiopaque. The Y-shaped portion of the cartilage separating ilium, ischium and pubis is much lessened. The epiphysis of the head of the femur becomes more dense, showing radiopaque areas, indicating more bone-forming activity. In a number of cases, a new, small ossification center appears at the very peak of the great trochanter. The diaphyseal lipping of the anterior inferior iliac spine shows

Fig. 10. Normal hips at nine years of age.

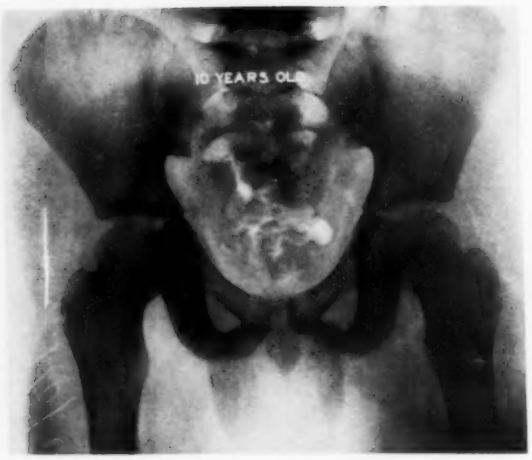


Fig. 11. Normal hips at ten years of age.

Fig. 13. Normal hips at twelve years of age. Note the first appearance of the new accessory rim epiphysis.

quite distinctly above the well-developed rim of the acetabulum. This started from a very slight projection first observed in the fifth year.

At eleven years (Fig. 12), there is a distinct change. The ossification centers are more active and the Y-shaped cartilage is beginning to show changes in its ossification. The bony elements of the acetabulum increase and the epiphyses are further advanced.

At the twelfth year (Fig. 13), we see an additional indication of growth. The coarse trabeculation of bone and the deposition of calcium out into the cartilage of the acetabulum to a separate ossification center, now appearing on the superior rim of the acetabulum, is definitely observed. Increased density, starting at the junction of the pubis with the ilium and extending throughout the anterior superior margin of the acetabulum, is noticed. This is, in my opinion, the anlage of a new epiphysis. The outermost margin of the anterior inferior iliac spine begins to appear radiopaque, as if some new activity were about to occur. The great trochanter has reached normal shape and its epiphyseal cartilage is narrowing. The epiphyseal cartilage of the head of the femur is narrowed and more completely ossified. The same

Fig. 12. Normal hips at eleven years of age.

Fig. 14. Normal hips at thirteen years of age. The new epiphysis has become clearly demonstrated.

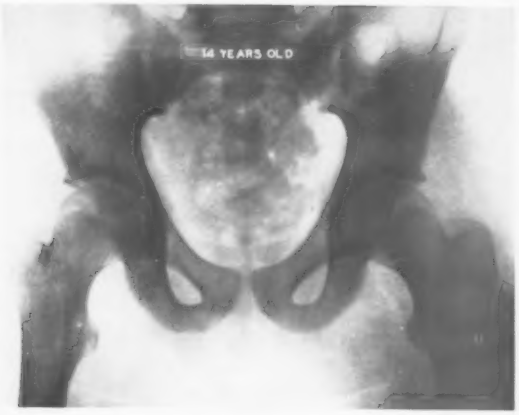


Fig. 15. Normal hips at fourteen years of age. Accessory epiphysis still present. Note how these new genetic centers have widened the acetabula.

process is evident in the lesser trochanter. The separate epiphysis, that is occasionally found, formed at the tenth year on top of the great trochanter, has now become a typical bony projection elongating the great trochanter.

At thirteen years (Fig. 14), the new epiphysis is advancing and a new outer acetabular rim can be determined. In the third year, it was noted that the acetabulum appeared formed as far as its superior lateral dimensions were concerned. From this age to the age of twelve, the growth of the femoral head has outstripped that of

Fig. 16. Normal hips at fifteen years of age. Accessory epiphysis still clearly shown widening the superior aspects of the acetabula. Conjugal cartilage as yet has not fused.

Fig. 17. Normal hips at sixteen years of age. Accessory epiphyses have fused with diaphyses to complete the superior acetabular rim.

the superior lateral acetabulum. At twelve, the head of the femur in some cases extends farther laterally than its acetabular bearing surfaces. It is to compensate for this more rapid growth that this new outer acetabular rim is formed with its new epiphysis. It is also necessary to round out completely the acetabular lip which bars the joint from dislocation in this direction. This new epiphysis, as before stated, may take the form of one or more ossification centers along the superior acetabular border.

The os acetabulum, which is the name given the portion of the Y-shaped dividing cartilage found between the ilium and pubis, has a very distinct ossification center. Ossification is occurring, as proved by

Fig. 18. Normal hips at seventeen years of age.

Fig. 19. Normal hips at eighteen years of age. Acetabular margins have well-developed lips. The epiphyses of the major and minor trochanters are completely ossified. Femoral capital epiphyses still unfused. Acetabular epiphyses, and those of the anterior inferior iliac spines are completely ossified.

the coarsened trabeculation, the radiopaque and the radiolucent areas around the iliac portion of the acetabulum. This appearance of growth activity is very evident when comparison is made with the portions of the acetabulum made up of ischium and pubis. Here growth is either very much slowed or stopped altogether. It will be noted that in the normal hip the acetabular portion, derived from the ischium and pubis, changes very little by lateral growth

Fig. 20. Case W at twelve years of age, with a tuberculous hip. Ossification center of epiphysis first appears on the left acetabular rim.

throughout its development, compared to the doubling or trebling in size which occurs in the iliac portion. At least part credit for this latter growth must be given to this new ossification center which is instrumental in forming the anterior superior, superior and posterior superior portions of the acetabular rim. The epiphysis of the head of the femur and great and lesser trochanters at this age show more complete ossification.

At fourteen years (Fig. 15), the ossification of the head of the femur and lesser and greater trochanters is more distinctly seen.

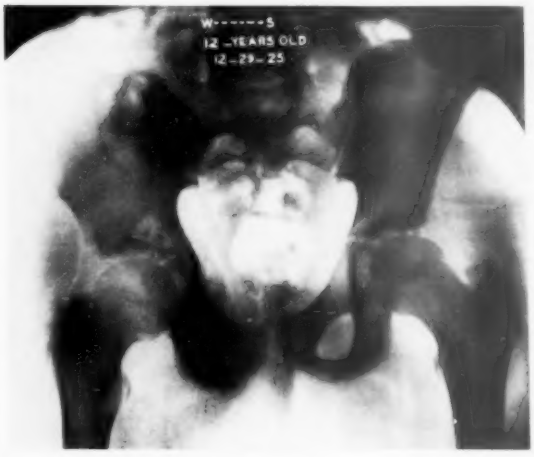


Fig. 21. Case W at twelve years, three months—ossification center of epiphysis on left increases, one on right first appears.

The acetabular rim shows an increase in size of the ossification centers newly established on the outer rim of the acetabulum. The anterior inferior spine seems to project farther with considerable radiolucency on its outer margin.

At the fifteenth year (Fig. 16 and 17), the epiphyseal centers are all advancing and all the bones are increasing in size.

By the seventeenth year (Fig. 18), the acetabular rim is completely formed. The epiphyses of the major and minor trochanters are closed late in this year. Only a line can be seen at the head of the femur at its junction with the neck. The acetabular rim still shows its smooth outline with evidence of bony activity in its continued density.

The epiphysis of the anterior inferior iliac spine may close from the sixteenth to the eighteenth year. The acetabulum is now complete (Fig. 19).

The first appearance and fusion of all epiphyses are again quite variable. This seems dependent on general well-being or endocrine balance. The earliest sign of ossification of the acetabular rim I have discovered was at eleven years; while the latest complete fusion was in the seventeenth. Growth of bony tissue is indicated by a radiopacity, coarse-textured structure or trabeculation giving a roughened appearance to an otherwise smooth contour. This will be demonstrated in two cases followed by a series of roentgenograms through their growing period.

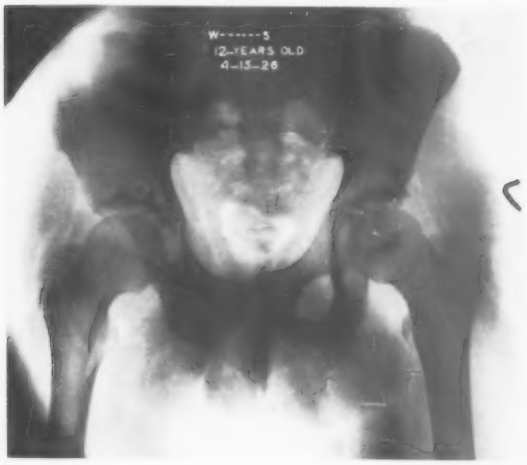


Fig. 22. Case W at twelve years, seven months—ossification centers of epiphyses of both acetabular rims are distinct.

That this variation in depth of the acetabulum depends not only on the epiphyseal growth of the conjugal cartilage between the ilium, ischium and pubic ramus, but also from this decidedly separate genetic center on the superior acetabular rim located principally in the ilium, must not be overlooked. It is this center which normally, between the ages of ten to twelve years, forms an epiphysis which fuses between the ages of seventeen to eighteen to complete the superior acetabular rim.

Fig. 23. Case W at twelve years, eleven months—accessory rim epiphyses gain in size.

In order that the development of these accessory acetabular rims may be completely understood, as far as our knowledge of today allows, I wish to present two cases which were followed through this period with a series of roentgenograms.

First, I wish to emphasize a few of the salient facts. About the eleventh or twelfth year, the acetabulum is well developed. The ragged character of the bone of ten years is beginning to give way to a smooth appearance. However, there are at this time quite numerous areas of bone buds with accompanying radiolucent areas of cartilage. The large V-shaped notch seen

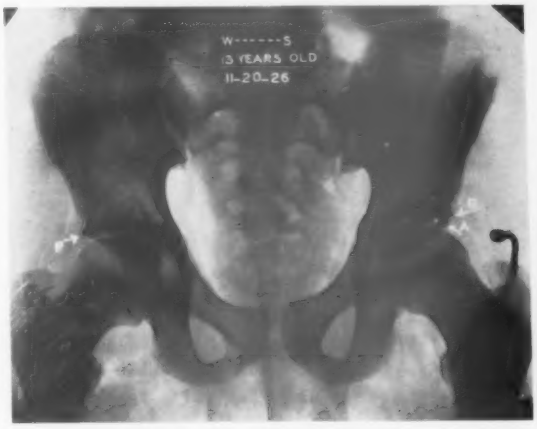


Fig. 24. Case W at thirteen years, two months—left acetabular rim shows two centers of ossification to one on the right which has become smooth.

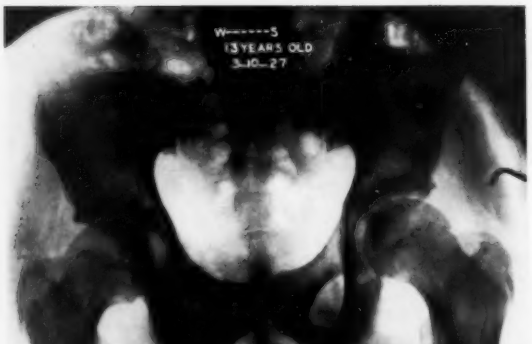


Fig. 25. Case W at thirteen years, six months. Complete union of epiphysis on right acetabular rim.

originally between ilium and ischium is now nearly filled with bone and shows ossifying activity. The femoral head has at this time a little more than filled the bony acetabulum. To widen and deepen the cavity, a new ossification center appears on the present superior acetabular margin. As many as three distinct centers may establish themselves—one anterior superior, one superior, and one posterior superior—all extending along the rim of the acetabulum and originating mostly on its iliac portion. In this series of roentgenograms taken on patient W, we start with him at the age of twelve.

Fig. 26. Case W at thirteen years, nine months. Fusion of epiphysis on left acetabular rim.

Age twelve (Fig. 20). The ossification center is observed on the left superior acetabular rim and the epiphysis of the great trochanter is well outlined. The epiphysis of the femoral head is dense and the trabeculation of the acetabulum is completed except for its rim.

Age twelve years, three months (Fig. 21). The ossification center has increased in size over the superior acetabular margin and a definite ossification center is now seen to have appeared on the right superior acetabular rim. There has been an advance in the epiphysis of the lesser trochanter. Again the coarse trabeculation, an index of bone growth, indicates that more bone is being developed around the acetabulum and practically all this activity is restricted to its anterior, superior and posterior rim margins, which are now becoming more dense.

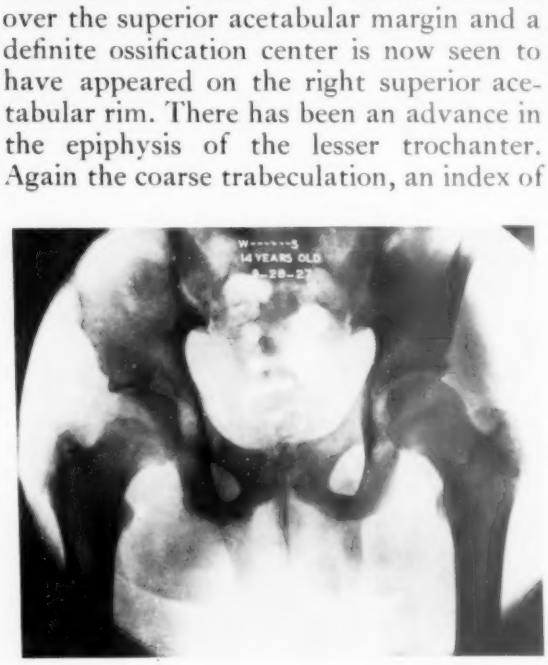


Fig. 27. Case W at fourteen years. Both acetabular rims are completed and project over a quarter of an inch beyond the outline of the plates taken three months earlier.

Age twelve years, seven months (Fig. 22). The epiphyses of the acetabular rim on both sides are more distinct. The rim is beginning to project laterally. The right acetabulum is developing somewhat more rapidly, because of the tuberculous area in its rim just above the epiphysis. Both the greater and lesser trochanters show an increased development and the epiphysis

of the head of the femur is becoming more shallow. The trabeculation is more pronounced throughout the acetabulum, denoting active bone growth.

Age twelve years, eleven months (Fig. 23). The projection of the superior lip of the acetabulum is more distinct. The projection continues as the epiphyses advance. This picture in stereo shows one center on the posterior margin and one on the superior margin of the left acetabulum.

Age thirteen years, two months (Fig. 24). The epiphysis on the right acetabulum is quite smooth on its posterior superior aspect. The trabeculation over the anterior

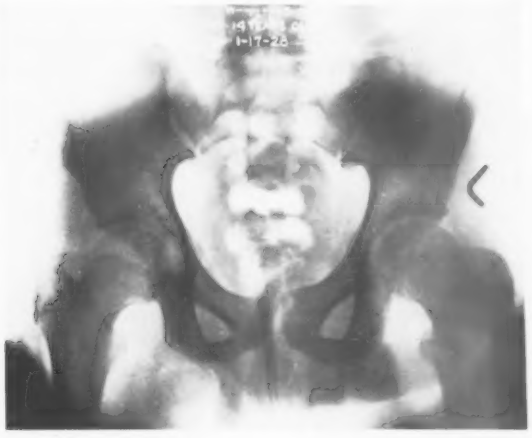


Fig. 28. Case W at fourteen years, four months.

superior portion of the acetabulum is distinct and gives a mottled appearance. The left acetabular rim shows two centers of ossification. The epiphyses of the greater and lesser trochanters are closing.

Age thirteen years, six months (Fig. 25). There is complete union of the epiphysis on the right acetabulum but not complete fusion on the left acetabulum. The acetabula still show evidence of growth activity. The epiphyses of the upper ends of the femurs are advancing. The acetabular margin is beginning to become smooth in appearance.

Age thirteen years, nine months (Fig. 26). The right hip shows a fairly smooth posterior and superior ridge of the outer acetabular lip. The left hip shows a well-

Fig. 29. Case W at fifteen years, three months. Right hip melting away from the tuberculous process.

formed acetabular lip. There is complete closure of the epiphyses between the ilia, ischia and pubes, although their outline may be made out. The major and minor trochanters show their epiphyses advancing.

Age fourteen years (Fig. 27). Both articular rims of the acetabula are developed, projecting nearly a quarter of an inch beyond the outline of the plates taken three years earlier. The bone now has a more nearly radiopaque appearance. The trabeculations appear more regular, comparable to adult bone. The epiphyses of the major and minor trochanters are both slowly disappearing.

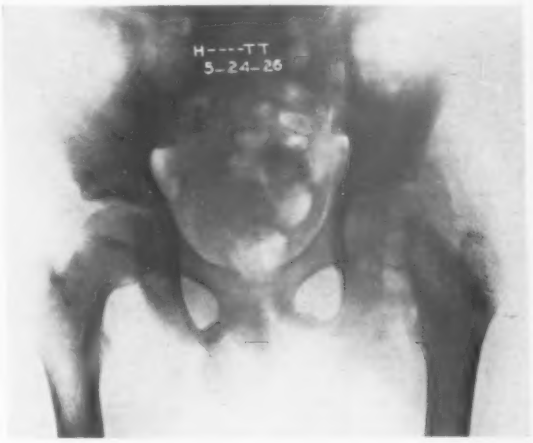


Fig. 30. Case H, aged twelve. Patient has multiple osteomyelitis involving the neck of the left femur.

Fig. 31. Case H, aged twelve years, two months.

Age fourteen years, four months (Fig. 28). The acetabular outlines are well developed. The coarse trabeculations of the growing bone centers have become normal like the adjoining bone with the exception of the anterior superior area. There is a definite lip on the fully formed acetabulum. The epiphyses of the acetabula are completely ossified. The epiphyses of the major and minor trochanters and heads of both femurs are nearly ossified, just a line being visible.

Age fifteen years, three months (Fig. 29). The left hip shows complete ossification of the epiphyseal line of the major trochanter,

Fig. 32. Case H, aged thirteen years, one month.

but not quite so complete ossification as that of the lesser trochanter. The cartilage line can be seen. The bony construction of the acetabulum is completed with just an area of density on its superior margin. The right femoral head is beginning to melt away with a tuberculous process.

A series of roentgenograms were taken on a boy, Case H, beginning at the age of twelve.

Age twelve years (Fig. 30). He has multiple osteomyelitis, one area of which involves the neck of the left femur. The contours of the acetabula show what appears to be normally fused superior margins. The os acetabula are just beginning to show.

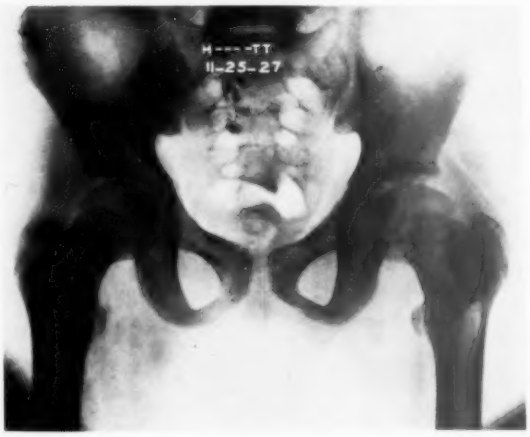


Fig. 33. Case H, aged thirteen years, six months. Accessory epiphyseal rim first appears on left acetabulum.

The left acetabulum is slightly further advanced, as would be expected when there is either an inflammatory or traumatic process. The cartilage between the ilium and ischium still shows as quite a wide space. The acetabular rim appears dense and calcified from the bony centers that are developed.

Age twelve years, two months (Fig. 31). A slight advance in the ossification processes is observed. The ossifying trabeculations are more distinct on the left than on the right. The epiphyses are more advanced and the lesser trochanters show quite distinctly.

Age thirteen years, one month (Fig. 32). The ossifying trabeculations have increased, filling in the cartilage and deepening the acetabula. The greater bone activity on the left hip over that of the right is very marked.

Age thirteen years, six months (Fig. 33). The epiphyseal spaces are much greater and begin to show an accessory ossification center on the left posterior superior acetabular rim. No similar process is discernible on the right. All the epiphyses show increasing activity. The epiphysis of the head of the left femur is practically fused. The left acetabulum is flattening to conform to the flattened femoral head.

Fig. 34. Case H, aged fourteen years, one month.

Age fourteen years, one month (Fig. 34). A line of demarcation for this left accessory acetabular rim appears as the character of these epiphyses becomes more definite. The femoral head and acetabulum are well flattened. The right acetabulum is not developing as rapidly. The accessory epiphyses are still absent on the right.

Age fourteen years, five months (Fig. 35). The epiphyses of the major and minor trochanters are nearly completely fused. The epiphyseal line of the left acetabular rim is still in evidence. The ossification centers in the cartilage of the acetabular cavity are now accentuated, denoting bone activity.

Age fourteen years, eight months (Fig. 36). The outline of the left acetabulum is nearly completed. The flattening process continues. The epiphysis of the os acetabuli on this side is nearly closed. The right acetabulum is just beginning to present calcified structures in its development. The ossification of the right posterior acetabular rim is for the first time visible as a faint epiphysis appears on the posterior superior acetabular rim.

Fig. 35. Case H, aged fourteen years, five months.

Age fifteen years, one month (Fig. 37). Bone trabeculations are still in evidence in the left acetabulum. The right acetabulum presents distinctly the accessory epiphysis

Fig. 36. Case H, aged fourteen years, eight months. Right acetabulum first shows accessory rim epiphysis.

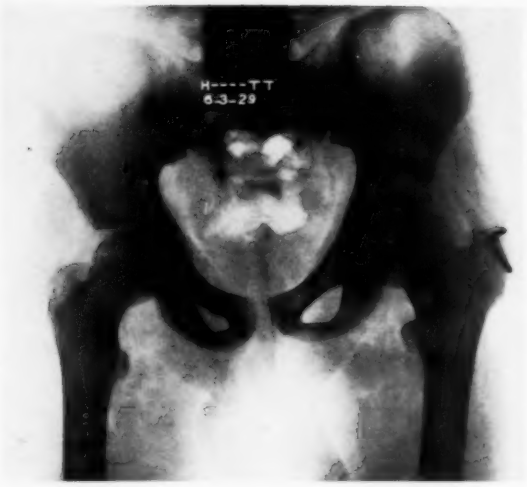


Fig. 37. Case H, aged fifteen years, one month. Right acetabular accessory epiphysis is distinct.

and area of ossification. The ilium, ischium and pubis are nearly completely fused in in both hips.

Age fifteen years, three months (Fig. 38). The epiphyses are closing in and the trabeculations are less coarse on the right side. The left acetabulum presents a flattened margin surrounding a large, flattened femoral head. The epiphyses of the femoral head and both major and minor trochanters on the left are practically complete, while those on the right side are still in evidence.

Age sixteen years (Fig. 39). The left acetabulum presents its final shape which is flattened to conform with the flattened head of the femur. Its contour is smooth and the trabeculations become finer. There is almost complete fusion of the major and minor trochanters. Processes of ossification on the right are distinctly seen in the superior and posterior margins. The acetabular rim is forming and demonstrates a clear epiphyseal line.

In this case we have two processes occurring, one which slows growth and one which speeds growth. In the first place, the patient's general health was such as to retard the beginning formation of the acetabulum from twelve to fourteen years of age on the right hip which was normal. The left hip, which had the same retardation factor, had, besides this, an inflammatory process in the form of multiple osteomyelitis, one area of which involved the upper end of the femur. Poland, in his book, "Traumatic Separation of the Epiphyses," quotes Ollier as saying:

> The earlier junction of an epiphysis is the index of an exhausted nutrition, that the epiphysis joins on first where the increase in growth has almost entirely ceased, and that the end which joins on last is that which has the greatest activity in proliferation; this difference between the two ends of a bone being manifest from the time of birth and it may be before any of the epiphyses are joined.

This case seems to substantiate this law. The left hip with identical factors to retard its growth two years, through its inflammatory condition and thereby exhausted nutrition, is practically completed when its normal brother is just starting. This is quite a striking contrast and shows the numbers of factors which enter into normal bone formation

Case III (Fig. 40) is a plate taken of the pelvis of a boy of fourteen years. One immediately notices the dense trabeculations of all the pelvic bones, denoting the rapidity of growth. There are also well-defined epiphyses on the superior posterior margins of both acetabula. There is a distinct de-

Fig. 38. Case H, aged fifteen years, three months.

lineation between the epiphyses and diaphysis demonstrating the extent of growth required to form this outer lip. If a view could be taken through the anterior lip, we would find epiphyses here. Sometimes viewing stereoscopically shows this anterior epiphysis clearly. The os acetabula are fused. It is quite evident in this roentgenogram how much these epiphyses deepen the acetabulum and how necessary they are to the production of a normal hip socket.

In the observation of these three cases, one very interesting fact must be noted. We believe we have definitely proved that the acetabular rim has been completed by a new epiphysis arising from a genetic center on the superior acetabular margin which is composed mostly of the ilium. We must define two points in order to answer the question as to whether these rims are formed by an ossification center or by a true epiphysis.

An ossification center is a definite area in cartilage whereby bone is laid down to complete the formation of bone. This is done by sending out irregular periosteal buds into the cartilage. These grow in all directions, involving the cartilage irregularly throughout their extent. This is what I referred to when I described a trabeculated, roughened or ragged appearance.

An epiphysis, on the other hand, is a bone germinating center separated from a long bone by a line of cartilage which, after the bone has reached the limit of its development, disappears and allows the smaller element to fuse onto the larger.

Fig. 39. Case H, aged sixteen years. Left acetabulum complete. Right acetabular rim still forming.

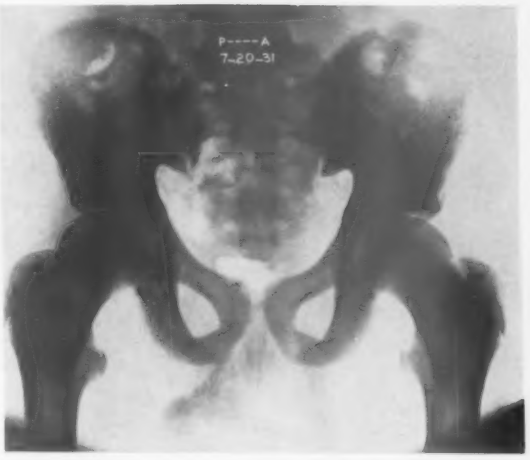


Fig. 40. Roentgenogram of fourteen year old showing the well-defined epiphyses on the superior rims of the acetabula, derived principally from ilia. Represents normal stage in growth, not fracture. Epiphysitis may develop here.

In following through the series, it is evident that these bony elements do not grow as was postulated for an ossification center. There is a definite layer of cartilage between the bones on what would be the diaphyseal side. This epiphysis, therefore, grows in a uniform way and keeps its definite line of cartilage about the same width over a period of months while shaping itself. At this point it fuses with the diaphysis just as the epiphyses do on the lesser trochanter, on the anterior superior iliac spine and the ischial tuberosity. From all this I am led to conclude that the elements with which we are dealing are true epiphyseal centers.

As an additional proof that these acetabular rim elements are true epiphyses, we frequently find ossicles placed on the anterior, superior and posterior acetabular rim. These serve to deepen the acetabular socket. It is well known that an epiphysis during its formative period may possess a conjugal cartilage which for some reason unknown may fail to ossify. This conjugal

Fig. 41. Note fleck of bone at arrow with radiolucent area at acetabular rim. This is cartilage and is frequently mistaken for a line of fracture or an arthritic change. It is an unjoined epiphysis or ossicle.

cartilage may undergo vacuolization and a separate joint lined with hyaline cartilage may form, separating the epiphysis from its diaphysis. Dr. Z. B. Adams, of Boston, in a personal communication, has described operating on one of these cases and removing an ossicle from the superior rim of the acetabulum which presented a smooth articulating surface.

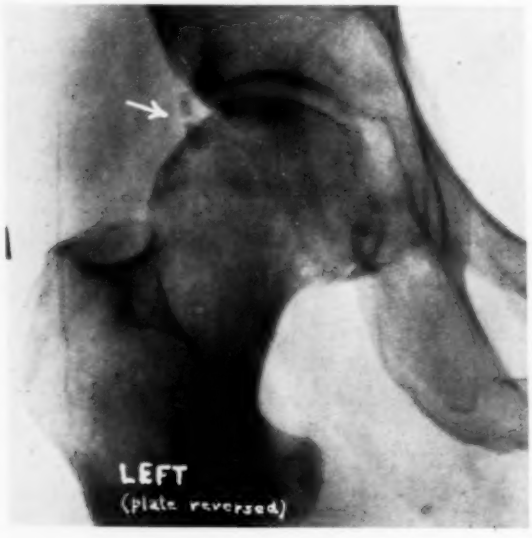


Fig. 42. Three separate ossicles, one on the superior and two on the posterior acetabular rim. They are separated by a radiolucent line of cartilage and tend to deepen the acetabulum.

Here we believe is found an explanation to account for ossicle formation on the superior acetabular rim. One of these accessory rim-forming epiphyses, which normally fuse about the seventeenth year, has for some reason or other lost its power to join its diaphysis due to a defective ossification of its conjugal cartilage. The latter, instead of ossifying, vacuolates and a joint is formed between the potential epiphysis and its diaphysis. This is not an unusual procedure with epiphyses. Examples of other ossicles resulting from non-fusion of

Fig. 43. Right hip shows a separate ossicle on the superior margin. It is dense, showing bony trabeculation, and is not homogeneous like a sesamoid. Left acetabular rim shows two ossicles on the superior margin separated from the rim by cartilage. Femoral heads are acorn-shaped, similar to coxa malum senilis.

epiphyses are numerous: on top of the great trochanter, on the anterior superior iliac spine, on the inner aspect of the navicular bone, on the end of the ulna styloid, on the lower end of the fibula, etc.

Meyer, at the end of his chapter quoted above, writes of these ossicles which persist after occlusion of growth: "Whether it is the result of broken down marginal eminences, abnormal ossification in the capsular appendage, arthritis deformans or a persisting acetabular wall nucleus, the pictures do not say." (They appear to be

definite ossicles with articular surfaces as described above and are due to incomplete development.)

Our studies have led us to conclude that an epiphysis may do one of three things. (*a*) It may fuse to its diaphysis in a normal manner. (*b*) It may remain separated from the diaphysis by a layer of cartilage which is the survival of the epiphyseal cartilage which has never ossified to join the epiphysis to diaphysis. (*c*) It may become an ossicle due to the vacuolization of its conjugal cartilage which disappears, becoming a joint cavity between the ossicle and its diaphysis. This development may not only

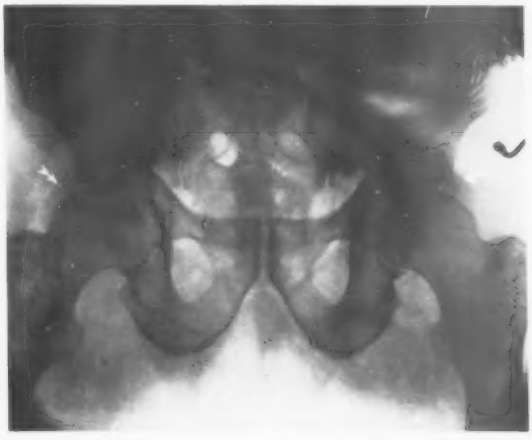


Fig. 44. Large separate ossicles on the posterior superior rims articulating with the ilia. They help to form the supporting joint cavity. In addition, arthritic changes are seen on the left.

stop at one of the arbitrary phases, but may stop at an intermediate or transitional type. Figures 41 and 42 demonstrate the second type of development. The cartilage in Figure 41 is still connecting the bony spicule to its diaphysis. This condition is often mistaken for bony overgrowth or some form of arthritis. It simply means that a portion of the cartilage has failed to ossify, leaving a bony center separated from the acetabular rim by cartilage. Figure 42 shows where three separate ossicle-like areas are discernible on the left acetabular rim. In this case the conjugal cartilage still remains between the bony center

Fig. 45. An elongated posterior superior rim on the right, having a distinct radiolucent line of cartilage. It deepens the posterior aspect of the acetabular rim. There is a small ossicle, likewise placed, on the left. These are congenital, not fractures.

and its diaphysis. This is additional proof of the essential rôle that the normal fusion of these bony elements has in the formation of the acetabular rim. Figures 43, 44, 45, 46 and 47 illustrate the third type of development. The conjugal cartilage is vacu-

Fig. 46. Osteitis deformans. A definite ossicle is seen deepening the acetabulum.

Fig. 47. Radiolucent area in the rim of the acetabulum in the outer epiphyseal portion. This is the most frequent seat of involvement in certain forms of arthritis. Degeneration starts here.

olized, leaving a joint cavity between the bony center and the diaphysis. Figure 43 shows two definite articulating ossicles, one each on the posterior rims of the right and left acetabula. The acetabula have deep superior margins.

Figure 44. This patient suffered pain in relation to the left hip and here we see a large articulating ossicle extending on either side of the superior posterior rims as

Fig. 48. Right hip shows the radiolucent area on the posterior superior margins of the right acetabulum, a congenital condition. The left hip presents a line of demarcation along the ischial portion of the acetabular rim. This is a fracture.

Fig. 49. There is a radiolucent line separating a bony area on the posterior superior aspect of the acetabulum. Diagnosed wrongly as fracture. It is congenital.

seen in the stereoscopic plates. These aid in deepening the acetabulum. On the left a definite arthritis is taking place in the articulation between the ossicle and the superior outer rim of what would be the diaphyseal portion of the acetabulum.

Figure 45 shows the deepened acetabular rim on the left posterior and superior margins. A separate ossicle-like area is observed on the posterior margin of the right rim. The cartilage has not fused and the

Fig. 50. Case of congenital separation of part of acetabular rim from diaphysis.

Fig. 51. Note the homogeneous appearing sesamoid under rectus femoris tendon. The acetabular rim is almost always irregular when a sesamoid is present.

rim, while projecting, has not completed itself as on the left.

Figure 46 shows a separate articulating ossicle over the right acetabulum in a patient who has Paget's disease with an ordinary type of coxa malum senilis (Fig. 47). Infection may develop early at the junction of the epiphysis with the diaphysis of the acetabular rims (Figs. 48, 49 and 50).

These acetabular ossicles must be differentiated from arthritic changes and, in case of injury, from fractures, especially in industrial medicine. There may be abnormal bony ridges with a thin area of cartilage between the outer periphery and main portion of the acetabulum and they are sometimes described as healed fractures. These are congenital malformations. In case of a suspected fracture line, a subsequent roentgenogram will show no line of demarcation, if a fracture existed.

Ossicles must be differentiated from sesamoids (Fig. 51). The latter are usually rounded and lack the bony structure characteristic of ossicles. There may be one or possibly two usually found on the superior margin of a poorly developed acetabulum. It was Dr. A. H. Pirie, of Montreal, who described these sesamoids with their relationship to the bursae under the deflected head of the rectus femoris tendon. They serve to take the pressure of the tendon from the poorly developed acetabulum. This position is characteristic and is a determining factor in the diagnosis. Closer examination will show a deficient and irregular acetabular rim. The sesamoid appears granular, differing from the bony trabeculation seen in the ossicle.

The presence of a second epiphysis on the great trochanter of the femur is sometimes

Fig. 52. Second epiphysis is starting on the tip of the great trochanter. Patient is nine years old.

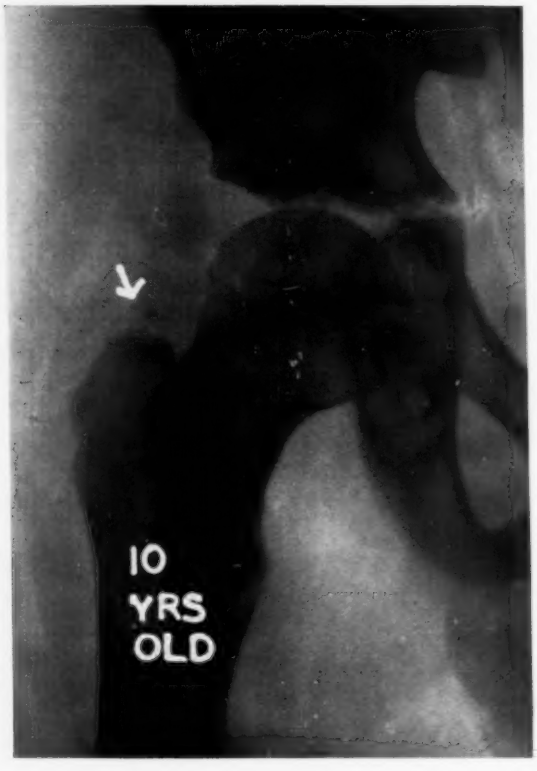


Fig. 53. Patient of ten years shows development of this secondary epiphysis on tip of great trochanter. Usual age for this epiphysis to appear.

noted at the ninth year (Figs. 52 and 53); but more frequently at the tenth year. Here again may be found a separate ossicle presenting a true articulation. This is caused by a congenital lack in the conjugal cartilage which fails to unite epiphysis with diaphysis (Figs. 54, 55, 56 and 57). This must be differentiated from a fracture. The ossicle presents a smooth base facing a likewise smooth adjacent portion of the trochanter. This condition remains unchanged, while a fracture will heal and the dividing line will disappear in later plates. In place of this second epiphysis a bursa (Fig. 58) may occur, causing pain and tenderness due either to infection or trauma. One, two or even three separate bursae have been observed (Figs. 59*a* and 59*b*). These appear to be of the same density as those observed in subdeltoid areas.

There is a third area on the hip where bursae may form (Fig. 60). This is about midway down the side of the great trochanter. This varies in different persons and it is especially noticed in certain infections where calcification first takes place, followed by fibrosis and finally terminated by ossification. The latter stage may be mistaken for a fracture. It is a large, irregular, exostosis-like process in place of bursae (Fig. 61).

I wish now to discuss very briefly the types of normal adult hips which are seen routinely. For convenience sake I shall tabulate these observations rather sketchily.

1. Variations in the Acetabulum (Figs. 62, 63 and 64).

a. The shape of the acetabulum as we normally see it is generally rounded with smooth margins. Its contour may vary towards the elliptical, especially on the anterior superior margins.

b. The depth of the acetabular cavity

Fig. 54. Greater trochanter is elongated due to the ossification of the second epiphysis.

Fig. 55. Small pointed bursa on the tip of great trochanter. Note its homogeneous character.

Fig. 56. Normal formation of great trochanter.

Fig. 57. Separate ossicle on the tip of adult great trochanter.

is a great variable. It may be extremely shallow, barely supporting the head of the femur, or it may be distinctly cuplike with a definite overhanging margin. The deep socket has a decided rim at that portion of the acetabulum developed from the ilium and the accessory ossification centers. The acetabular depth is proportional to the de-

Fig. 58. Note homogeneous structure of bursa on great trochanter.

Fig. 59*a*. Two bursal areas on the great trochanter with their homogeneous structure.

velopment of these accessory ossification centers and epiphyses in this portion of the acetabulum.

c. The acetabular position is usually described as pointing downward, forward and outward, depending considerably on the

Fig. 59*b*. Two bursal areas with their homogeneous structure found on the great trochanter.

Fig. 60. Bursal area low down on side of great trochanter. There is also a bursa on its superior margin.

tilt of the pelvis and upon the sex of the individual; varying the position of the pelvis when the plate is taken makes very little difference.

Fig. 61. Large area of calcifying bursa over the great trochanter extending into the soft parts adjoining.

Fig. 62. Rounded femoral head, shallow acetabulum, and a short femoral neck.

2. The anterior inferior iliac spine may be almost entirely absent, or it may form a considerable ossicle-like projection over the rim of the acetabulum (Figs. 65, 66 and 67), forming a definite union with and helping to form a portion of the rim of the acetabulum. In a number of cases it has been observed as a long projection almost articulating with the major trochanter (Figs. 68 and 69). Its epiphysis forms by the twelfth year and closes by the sixteenth to the seventeenth year.

3. Variations in the Femur.

a. The head may vary in shape. It is usually rounded and conforms to the acetabulum. If the acetabulum deepens, it may sometimes elongate and become pointed. It may become rounded and elongated in some chronic infection like Neisser's infection, described as coxa malum senilis (Fig. 70). It comes off at an oblique angle to the pelvis, and this obliquity has wide individual variations which are offset by accentuating the angle of the femoral neck. We consider the normal angle about 125° and all variations of obliquity between coxa vara and coxa valga are every-day occurrences (Figs. 71, 72 and 73).

b. The neck may be shortened or elongated. Its angle with the shaft varies considerably. "The angle of inclination is an angle formed by the axis of the head and neck with that of the shaft. It is about 125° in adults, slightly more in children." Fairbank continues: "The angle of ante-version or ante-torsion, which is the angle that the axis of the neck makes with the transcondylar line, shows even greater variation in the hips which display no instability. Pearson gives the mean figures of this age to be 15.3°. Burghard says it varies from 15° to 25°. Soutter and Bradford, in 154 normal femurs showed the angle varies from 37° to 35°—an average of 14.3°."

It must be borne in mind that the hip itself offers as many variations from any set normal as the individual as a whole does. There are small acetabula, large acetabula, narrow acetabula, wide acetabula, deep

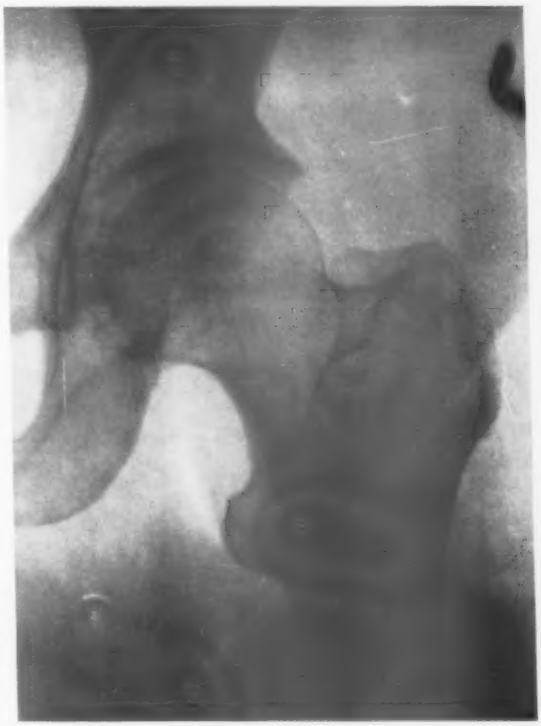


Fig. 63. Deep acetabulum posteriorly. Note shell-like rim.

Fig. 64. Moderately deep acetabulum, prominent anterior inferior iliac spine and separate ossicle posteriorly to deepen the cavity.

acetabula and shallow acetabula. There are infinite variations in the length of the neck of the femur, its angle with respect to the shaft and the size of the large trochanter and the bones themselves. What are the reasons for such variations? We are inclined to believe that such variations are just as much inherited characteristics as are color of eyes or shape of head. They are all factors of genetic laws governing our total makeup. This individuality must not be overlooked, and nowhere is it so evident as in a series of roentgenograms of different pelves. If certain genetic centers develop and their epiphyses join normally, we then find what we consider the normal hip. On the other hand, if for some reason unknown, possibly a slip in the genes somewhere, these genetic centers either fail to develop altogether or, if they do develop, fail to join normally, then we have a picture which suggests the congenital hip. One needs to be very careful in making diagnoses unless congenital variations can be ruled out.

Fig. 65. Congenital variation in anterior inferior iliac spine.

Fig. 66. Variation in anterior inferior iliac spines. This aids in the formation of the acetabular rims.

In reviewing the literature on congenital hips, we find many ingenious though con-

flicting articles which seek to account for this anomaly.

Le Damany, in his book, "La luxation congenital de la hanche," believes that the congenital hip is due to position and pressure exerted in utero at its articulation.

Jansen, in his article on hip anomalies, quotes Le Damany:

> Even in the foetal state man differs from the animals through the large circumference of the pelvis. When pressure is exerted against the knees of the foetus, while the hip joint is flexed, the wide pelvic girdle in the region of the anterior superior spine offers to the femur a fulcrum more easily in man than in the animals, so that the head of the femur is levered out of the hip socket.
>
> The floor of the socket, which is not any longer acted upon by the pressure of the femoral head, remains too thick; the socket itself consequently becomes too shallow; and in most serious cases dislocation of the hip ensues. Even normally the wide pelvic girdle is the cause of an increase in shallowness of the hip

Fig. 68. Tongue-like projection of anterior inferior iliac spine extending downward over head and neck of femur.

Fig. 67. Variation in anterior inferior iliac spine helping to form the posterior superior rim of the acetabulum.

socket during the last three months of prenatal life. After birth, however, it becomes deeper again. During the first six months of prenatal life, the socket resembles a half globe, and during the last three months it diminishes in depth from 0.5 to 0.4 times the diameter of the socket. Hence, congenital hip dislocation is simply an accentuation of a phenomenon which occurs normally in the development of every human foetus. It occurs when the coverings of the foetus are smaller than they should be. Then the thigh is flexed in the hip and the femoral head is levered out of its socket more forcibly than in the normal condition, the wide pelvic girdle offering a fulcrum. In this mechanical action the length of the human femur, which exceeds that of animals, plays a part, enabling the lever action to take place with relatively great force. The fact that the female foetus has a wider pelvic girdle than the male is in accordance with the fact that women are more often affected by congenital hip dislocation than men; and the reason why . . . the animals are exempt from congenital hip dislocation is found in the fact that they have a smaller brain, a comparatively

Fig. 69. Normal epiphysis of the anterior inferior iliac spine.

smaller head and a relatively narrow pelvic girdle.

Jansen agrees with Le Damany that the wide pelvic girdle and the large brain are the cause of congenital dislocation.

In our studies of many thousands of hips, a great many with congenital dislocations, we are convinced that changes in shape of

Fig. 70. Left hip presents normal acetabulum, normal head of femur and normal angle of inclination. Right hip shows rounded, elongated head with early production, and eburnation beginning coxa malum senilis.

acetabula during fetal life are the normal changes of growth, similar to the process we have outlined from birth to adult life. If one recalls physics and particularly the pressure of a fluid on its container or on any body immersed in it, he cannot help see, in the case of a child in utero, held in its membranes and entirely surrounded by fluid, that pressure must be equal in all directions over the surface of the embryo. The pressure should be as great on the great tro-

Fig. 71. Normal acetabulum, short femoral head with elongated neck.

chanter over the buttocks as on the end of the femur crowding towards the hip. That the child does not maintain its position throughout its life in utero is seen in numerous roentgenograms where the leg may be straightened to a right angle with the body, or even to a greater angle. In fact, the fetus constantly changes its position in utero.

Berry infers that there has been a partial arrest of growth at the reptilian stage of development, and this results in the imper-

fectly formed, shallow acetabulum and the malformation of the head of the femur. He maintains that with this arrest in development, congenital dislocation occurs when rotation of the limb takes place, in order to make the erect attitude possible. He quotes as one who believes that "Malformations of the human acetabulum correspond to forms found in lower animals," and like Lorenz, believes that "Congenital dislocation of the hip is due to arrested development." Berry concludes that: "The epiphyses found in reptiles are not always bony; but are sometimes merely calcified cartilages. It would follow, therefore, that the human hip joint that had been partially arrested in development at the reptilian stage would have a poor quality of epiphysis."

Farrell, Von Lackum and Smith, in a study of 310 cases, conclude that "acute version of the neck of the femur causing torsion is a prime factor in producing congenital dislocation. The femoral head protrudes from the front of the acetabulum and

Fig. 72. Coxa valga.

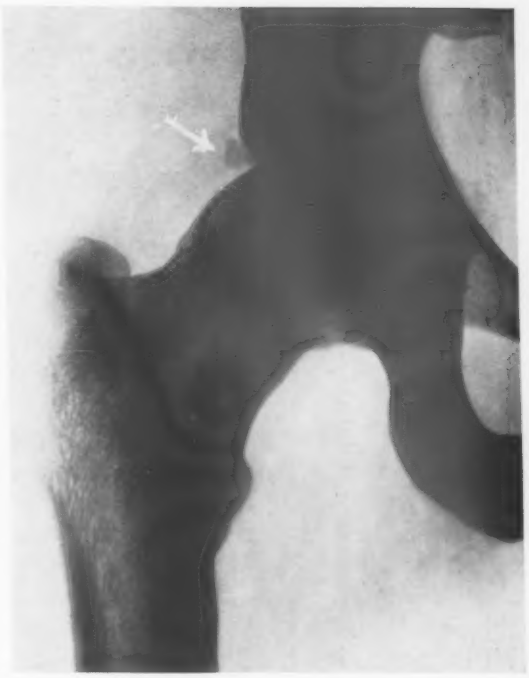


Fig. 73. Shallow acetabulum, two ossicles, elongated head and normal neck.

contracture of the cotyloid ligaments prevent replacement."

The cause of congenital dislocation of the hip is today a moot problem. Fairbank, in his Lady Jones Lecture delivered before the University of Liverpool, states:

As the result of his experience, if foetal specimens be examined, the most striking and often the only feature besides the slight displacement of the head of the femur is the poor development of the acetabulum. This is the primary fault to which all other changes are secondary. . . . Often after both forms of reduction, the acetabular margins must grow out again. Failure of the upper lips of the acetabulum to grow out seems to be a more potent factor leading to dislocation.

Willard echoes this in his article.

The X-Rays before reduction show the usual misplaced femoral head and the shallow socket with little or no deformity of the femoral neck or head. The X-Rays after reduction show a considerable deformity of the head and neck of the femur. The acetabula are shallow and of abnormal length. The widening is always in the upward direction and at times the socket has

over twice the diameter of the normal case. The upper rim is deficient, giving very poor support to the femoral head which lies in this upper portion of this socket. This deformation begins slowly after reduction.

Broca adds some significant figures. "In a study of the end result of congenital dislocation of the hip, perfect reconstruction of the joint is found in only twelve cases."

The explanation for these above observations lies in a complete understanding of the genetic center present in the upper acetabular rim. This center originates principally in the ilium. We cannot emphasize too much that the growth of the acetabulum is not entirely dependent upon the epiphysis located in the Y-cartilage between the ilium, ischium and pubis. Its widening and deepening is cared for by another genetic center which we mentioned as located principally in the ilium at the superior acetabular rim. Study of the normally developed acetabulum has demonstrated that long after this Y-shaped epiphysis has entered into the development of the acetabulum as far as possible, further deepening and widening is performed by means of this second genetic center. This new center continues the growth and normally develops definite epiphyses which enter into the formation

Fig. 74. Unilateral congenital hip for comparison of widths of the two acetabula at superior rim. Note that the right is twice the width of that of the left at the point marked.

Fig. 75. Congenital hips of child aged two+. Note position of femoral heads in relation to the epiphyses of the ilia and pubes. Pressure against lines of stress is causing absorption of the superior margins of the acetabula near the anterior inferior iliac spines. Genetic centers are undoubtedly present, and an early reduction would give a perfect result.

of the final superior acetabular rim as has been described. Absence of this genetic center explains the existence of the infantile or shallow acetabulum. It would seem that the transitional stages between a normal type hip and a congenital hip could be ascribed to the variations in the potential growth powers of this individual genetic center. In development, the acetabulum may be somewhat deepened by the stimulating pressure of a normal femoral head. This does not seem to be more than a minor factor, however, in acetabular formation. "Bones seem to have the power of resisting constant pressure in their longitudinal direction better than in the direction at right angles to it," to quote Jansen, in his "Bone Formation." In the acetabulum we assume that the bones, having been formed in this position, are built with pressure lines which do not conflict. Jansen continues, "Hence in the direction for which a bone is built any other non-functional pressure, be it constant or remittant, is withstood better than in directions at right angles to it."

In studying cases with unilateral congenital hip, it was first noted that the congenital hip acetabulum and surrounding bone grew only by uniform enlargement and was an approximating reproduction of the shallow infantile hip, only larger. On the other hand, the normal hip of the same individual, if followed over a course of years showed an alteration in general shape, and was not merely an enlarged infantile hip but of entirely different contour. The change in size and shape was found to occur on the anterior, superior and posterior portions of the acetabular rim. Measurements of the two acetabula in the adult were taken. In the inferior portion of both congenital and normal acetabula there was practically no difference in width. However, the upper portion of the normal acetabulum had increased to a width twice the same portion of the acetabulum of the congenital hip. If early pressure determines the depth of the acetabulum, why is there the near agreement in width of the lower part of the acetabula of both normal and abnormal hips and yet no agreement in the width of the iliac portions of these rims? Pressure will not account for all growth above the Y-epiphysis, nor will it explain the fact that the inferior portions of the acetabulum where pressure is exerted equally as great as on its superior portions because of its ball and socket construction, are nearly equal in width in both normal and abnormal hips.

Fig. 76. Double congenital hips with high false secondary joints. Superior lips of acetabula are deficient.

Most of the superior surface of the acetabulum is developed from the portion of the ilium that joins the ischium and pubis. In our study of congenital hips it has been this portion of the bone which has been found defective. We believe that it is not a pressure defect or the result of pressure applied upon the fetus in utero, but a congenital failure of these very essential rim genetic centers to develop, that is to blame for the pure type of congenital hip.

In Figure 74 we have a normal right hip while the left shows congenital dislocation. Comparison of the width of the two acetabula at the lower margins below the ischial spine show that they are nearly equal except for the normal diminution of the innominate bone due to loss of function from the abnormality. On the left, the outline of the acetabulum is visible. The anterior inferior iliac spine is obliterated by pressure of the dislocated femoral head. The width through this superior portion of the acetabulum on the left is about one-half that of the right, measured at the same relative position. This demonstrates how slight an attempt has been made to form the acetabular rim on the anterior or superior margins. The Y-epiphysis between the ilium, ischium and pubic ramus is completely fused on the right as on the left. This shows that the development of this Y-epiphysis increases the width of the acetabulum and has but little to do with the development of the rim (Fig. 75).

Figure 76. Patient is fifteen years of age. The anlage of the anterior, inferior spines of the ilia on both sides are evident. The dislocations are high; and false, secondary joints have been formed by nature when the genetic centers failed to construct a superior acetabular rim capable of holding the hip in position. That portion of the acetabular rim formed from the ilium and ischium can be seen.

In following many cases of congenital hip with series of plates through these vital ages, we have never been able to find the

slightest attempt to form the epiphysis at the superior rim of the acetabulum. The ossification is confined to the Y-shaped epiphysis between the ilium, ischium and pubis. This area of the acetabulum is found to develop much as it does in a normal child varying slightly to changing pressure. Closure takes place apparently a trifle sooner on the congenital side, as predicted by Ollier's statement on speed of epiphyseal development given above. The diminution in normal structures of the femoral head in the acetabulum decreases nutrition.

Figure 77. Patient was a rather large girl, aged fifteen, with elongated acetabula, especially on the right. The ossification centers of the major and minor trochanters, head of the femurs and the Y-shaped cartilage between ilium, ischium and pubis are all closed. There is a subluxation of the right hip and a partial subluxation of the left, the left acetabulum being very shallow, having a genetic center but no epiphysis. Note the shape of the femoral necks and heads, the anterior inferior iliac spine areas and the shape of the acetabula. Slight attempt has been made to develop the acetabular rim resulting from normal pressure on the left. There is no reconstruction on the right. The superior margin is ab-

Fig. 77. Congenital hips, partial on left, complete on right. Acetabula are very shallow. No secondary genetic centers present.

sorbed by lateral pressure, as is the region of the anterior inferior iliac spine; there are signs of regeneration of the bone to form a false joint. The first process noted is bone destruction. This is followed by bone regeneration since the pressure is intermittent and against lines of pressure.

Figure 78*a*. Girl, aged nine, with a normal right hip and complete dislocation of

Fig. 78*a*. Girl, aged nine, with normal right hip and co npletely dislocated left hip.

the left hip. The epiphyseal lines are in about the same stages of development, the left hip being slightly farther advanced. There is no attempt to form a left superior acetabular rim.

Figure 78*b*. Same patient at age of fourteen years. The acetabulum on the right is completed. Complete fusion has occurred on the left. Compare the widths of the superior and inferior portions of the two acetabular rims. Allowing for the deficient growth due to lessened nutrition, we still can see no attempt to form an acetabular rim.

Figure 78*c* shows only increase of growth with age. A false socket is well developed. Patient's age, fifteen.

The small percentage of complete surgical cures in the reduction of congenital hips in which the head of the femur remains in its true acetabulum are accounted for in the study of roentgenograms and are almost predictable. These operations were performed on hips which just approached the minimum in acetabular growth. That is to say, the superior acetabular rim possessed a genetic center deficient in epiphysis. The subsequent trauma and bone over-

growth nearly compensate for the deficiency in the genetic center. Comparison of the hips of these cases with those hips in what we believe to be a series of year by

Fig. 78*b*. Same patient at the age of fourteen. Right acetabulum is complete. There is no attempt on the left to build on the superior acetabular lip. Congenital lack of genetic centers.

year development shows that that portion of the superior acetabulum contributed by the ilium never exceeds that of a normal ten year old. The closer the approximation to this minimum and the earlier the reduction is performed, the more likely is the surgeon of success, since the cartilage reacts to form a bony ridge. In what we consider the true type of congenital hip which continues to redislocate after reduction, the picture is striking. The acetabula may range anywhere in development from the type of an infant at birth up to ten years. The worst dislocations are found in the type of hip normally presented at birth with the acetabulum slightly wider and more shallow.

When once a dislocated hip is returned to its normal position, we must not infer that the acetabulum deepens to any great extent. The growth which does take place is not just the result of the reduction, but of the potential power of growth retained in the genetic center. Pressure has little to do with this building process. There is doubtless a possibility that pressure may stimulate the genetic center to greater growth, but the normal potential of that genetic center is not exceeded. Jansen's law must be recalled concerning the withstanding of pressure by a bone built in the longitudinal direction of the strain. We are dealing with an acetabulum and a femoral head which are normally constructed to mutually withstand the pressure strains. After reducing a joint only a slight bony overgrowth may be expected, and this is referable directly to trauma, i.e., extra use directly applied. In other cases where a secondary joint has been formed, we find the answer in stresses applied against the normal pressure lines.

Bones are not made to withstand pressure at right angles to their normal pressure lines. Such cases show primary bone absorption and secondary productive changes finally yielding a makeshift secondary joint. This series of changes is observed in cases of true congenital hip: where the genetic center is absent and the dislocated head of the femur rests against the ilium

Fig. 78*c*. Same patient at the age of fifteen. Increase of growth with age. False socket on left is well developed. No sign of accessory superior acetabular rim element.

near the anterior inferior iliac spine. The irregular pressure causes first an absorption of bone, and secondarily a bone generation to form the false socket. This is the position in which most operable congenital hips are

found. The surgeon makes the superior rim posterior to and on a line with the anterior inferior iliac spine. His principal failures occur when reduction to this point is not

Fig. 79. Congenital hips have been traumatized in reduction. There has been no attempt by nature to develop the acetabular rims, although the femoral heads have been held in position. There is a slight traumatic overgrowth of bone seen in relation to the flattened heads. Above these are the traumatized anterior inferior iliac spines.

accomplished. In more complete dislocation the femoral head rests high up on the ilium, posterior to the anterior superior iliac spine and in this position generates its own joint. We cannot pride ourselves on a cure in these cases, since they result in Nature's handiwork and owe but little to our skill.

Contrary to the belief commonly held that the early congenital hip cannot be diagnosed by the roentgen ray in the infant, I believe that it can be diagnosed with certainty. The congenital hip socket at birth is long and shallow. That portion above the epiphysis is slightly elongated and much more shallow. The femoral head is still laid down only as cartilage, but stereoscopic plates taken of both hips with the legs extended and held in a proper position present this cartilaginous femoral head of the normal hip, below or near the level of the Y-epiphysis, while that of the congenital hip is shown in a higher position. The position of the head of the femur in the infant is determined by the diaphyseal portion of the epiphyseal edge of the femur.

Studies of series of roentgenograms taken during the period of epiphyseal development of any one joint will undoubtedly reveal many interesting and unsuspected variations. Secondary epiphyses, ossicles, fused ossicles, secondary joints at the sites of epiphyses, absence of epiphyses or diaphyses, accessory sesamoids and calcified bursae will be encountered by the roentgenologist and, especially in accident cases, must be differentiated from fractures. A few simple rules must be borne in mind in this connection. Epiphyses may fuse normally, remain ununited with intact conjugal cartilage, form an ossicle or by vacuolization of its cartilage produce a secondary joint. Bursae may calcify and later ossify, enlarging a bone. This structure differs from that observed in an injury. Knowledge of these abnormalities with their time and cause of formation and location will lead to a better understanding of many mechanical changes and defects in the skeleton which can be properly treated by the orthopedic surgeon.

Included in this study of the hip joint are the acetabulum, the anterior inferior iliac spine, the head of the femur, the femoral neck and the major and minor tro-

Fig. 80. High dislocation with coxa vara. Acetabula resemble those normally expected at birth. Early reduction would undoubtedly give good results, because observing width of iliac portion, it is evident that a genetic center is present sufficient to form a competent acetabulum.

chanters. Anatomists describe the acetabular region as presenting three primary and two secondary genetic centers of ossification. The former are concerned with the

ossification of the ilium, ischium and pubis respectively. One secondary center is concerned with the ossification of the anterior inferior iliac spine. The second is described as being "one or more" lying in the Y-shaped epiphysis, at the junction of the three main bones in the center of the acetabulum. The position of this latter, between ilium and pubis, is known as the "os acetabula."

In following series of plates taken over the growing period of the same individuals, we find an additional genetic center normally presented, which is most essential in the formation of a competent hip joint. It is located on the superior acetabular rim, principally in the ilium. About the twelfth year, this genetic center normally forms a true epiphysis on the superior acetabular margin. The epiphysis may divide into two or more ossification centers grouped around the anterior superior, superior and posterior superior acetabular rim. The possibility of this epiphysis has not been entirely overlooked; but its function and significance have never been understood.

This accessory rim epiphysis has a true epiphyseal or conjugal cartilage and like all epiphyses may follow one of three courses after its appearance at the twelfth to fourteenth year: (1) It may fuse normally to its diaphysis from the fourteenth to eighteenth year to accomplish the deepening of the acetabular cavity for the reception and retention of the femoral head. (2) It may remain separated from its diaphysis by a layer of cartilage, the survival of the epiphyseal cartilage which has never ossified to join epiphysis to diaphysis. (3) Finally, its conjugal cartilage may vacuolize and, in disappearing, leave a joint cavity between the bony element and its diaphysis. The latter is the origin of the congenital ossicle of the acetabular rim, so frequently found. This development need not stop at one of these arbitrary phases, but may show an intermediate or transitional type. That we are dealing with a true epiphysis is proved by: (1) it develops as a germinating center separated from bone by a line of cartilage which, after the bone has reached the limit of its development, disappears by progressive ossification allowing the smaller element to fuse onto the larger; (2) failure to fuse normally produces the common ossicle of the acetabular rim or a secondary joint, which must be differentiated from (*a*) arthritic changes, (*b*) fractures and (*c*) sesamoids.

In reviewing this we have included a complete anatomical description of the hip joint from the time of birth up to the eighteenth year, including a description of the appearance and development of the newly discovered acetabular epiphysis. In addition the different types of hip joints seen daily by the roentgenologist are described.

The congenital hip is seen to be the result of a retardation in the growth or a total lack of this very important genetic center. The configuration of a congenital hip is that normally presented by the child at birth, in the worst instances; or, at best, no further development than would be expected in a ten year old. Added to this picture is a slight elongation and flattening of the iliac portion of the acetabulum with the femoral head observed to lie above the level of the Y-cartilage. This demonstrates that the iliac portion of the acetabulum either lacks its genetic center on the rim, or possessing that, lacks the power to form the accessory rim epiphysis which at the twelfth year normally appears to enlarge the superior acetabular margin laterally.

In studying cases with a unilateral congenital hip, it was first noted that the congenital hip acetabulum and surrounding bone grew only by uniform enlargement and was an approximating reproduction of the shallow infantile hip, only larger. On the other hand, the normal hip joint of the same individual, if followed over a course of years, showed an alteration in general shape.

In all cases this was not merely an enlarged infantile hip joint but a joint of entirely different contour. The change in size and shape was found to occur on the an-

terior superior, superior and posterior superior portions of the acetabular rim. Measurements of both acetabula, in adults showing unilateral congenital hips, were taken. In the inferior portions of both congenital and normal acetabula there was practically no difference in width. However, the superior portion of the normal acetabula had increased in width to twice the size of the same portion of the acetabulum of the congenital hip.

Several outstanding theories as to the cause of congenital hip are presented from the literature and criticized from this standpoint.

Early congenital hips can be diagnosed by the following findings: (1) Long and shallow sockets; (2) that portion of the acetabulum above the epiphysis is slightly elongated and much more shallow; (3) the femoral head, judged by means of the upper end of the diaphysis, lies above the level of the Y-cartilage.

The constant finding of this accessory rim formation on the superior acetabular bearing surface can mean only one thing—it is vitally concerned with maintaining the joint's competence. Roentgenograms of congenital hips demonstrate these facts. Surgical prognosis is predictable by roentgen examination on this basis. We are forced to conclude that the accessory acetabular rim epiphysis, with its genetic center which has just now been described, is the foundation upon which our knowledge of the congenital hip and other anomalies of the hip should be based.

Acknowledgment is made to my son Harvey R. Morrison for his assistance in collecting references, arranging material and correcting proof.

REFERENCES

Berry, John M. Theory as to the cause of Perthes' disease based on roentgenologic findings. *J. Bone & Joint Surg.*, 1926, *8*, 333–347.

Broca, D'Antegnaca. *Rev. d'orthop.*, 1921, *3*, 353–357.

Fairbank, H. A. T. Congenital dislocation of the hip, with special reference to the anatomy. *Brit. J. Surg.*, 1930, *17*, 380–416.

Farrell, B. P., von Lackum, H. L., and Smith, A. de F. Congenital dislocation of hip. Report of 310 cases at New York Orthopoedic Dispensary and Hospital. *J. Bone & Joint Surg.*, 1926, *8*, 551–561.

Fromme, Albert. Die Bedeutung der Looserschen Umbauzonen für unser klinische Auffassung. *Arch. f. klin. Chir.*, 1921, *116*, 664.

Jansen, Murk. On Boneformation. Longmans, Green & Co., London, 1920.

Jansen, Murk. Large brain, wide pelvic girdle and outstanding number of hip anomalies in man. *J. Bone & Joint Surg.*, 1929, *11*, 461–531.

Le Damany, P. La luxation de la hanche. Felix Alcan, 1912.

Le Damany, P. La luxation congenital de la hanche. Flammesion, 1923.

Lilienthal. *Inaug. Diss.*, 1909, Königsberg.

Meyer, Hermann. Röntgendiagnostik in der Chirurgie und ihren Grenzgebieten. Julius Springer, Berlin, 1927.

Poland, John. Traumatic Separation of the Epiphyses. Smith, Elder & Co., London, 1898.

Rambaud, A., and Renault, C. Origin and Development des Os. Paris, 1864.

Willard, D. P. The results of the Davis method of reduction of congenital hip dislocation. *J. Bone & Joint Surg.*, 1927, *9*, 270–277.

DISCUSSION

Dr. Frederick W. O'Brien, Boston. I regret that the limitations of time didn't permit Dr. Morrison to read his paper. I have had the privilege of reading it several times and it is most informative and I think probably of very lasting value. Not only does it call our attention to the new centers along the superior rim of the acetabulum, but it gives us the reason why they are there—to deepen the rim.

Later when he discusses congenital dislocation, he infers that the reason we have congenital dislocation is because of the congenital absence of these new centers. His explanation, too, of epiphyseal changes, I think, is very illuminating and I am going to ask Dr. Morrison to differentiate between genetic centers and epiphyses, because I think in talking he usek them perhaps as though he believed they were the same thing, and yet I don't think he does. In reading his paper rather hurriedly, I tood

from it that in a practical way it will be of great value in industrial medicine. I know I have been guilty of sins of commission as well as omission in this regard. His explanation of epiphyseal changes, that is, the normal natural history, so to speak, of epiphyseal growth with union or non-union and the formation of not true joints and then perhaps later absorption or vacuolation of the epiphysis and ossicle formation and spur formation, means that we must differentiate between arthritic conditions and these congenital anomalies and fracture, especially in the industrial cases.

Dr. Percy Brown, Phoenix, Ariz. Dr. O'Brien took my initial words from my mouth when he said that the presentation time of this communication was much too short. I not only know what Dr. Morrison has said is but a part of his paper today, but also that he has the subject developed in other directions, so that, from what he tells me it would be quite likely that a monograph dealing with this subject will be published by him later on.

I can remember, as many of you can, the time when the hip joint was the criterion of our technical ability, from whatever source. The apparatus that could produce a portrayal of the hip joint within a reasonable time, anything within five minutes, was considered wonderful. In the brilliance of our later work on the large viscera, our old friend, the hip joint, has perhaps faded a bit into the background and has perhaps become neglected. One has only to read such communications as the last English translation of Dr. Alban Köhler to realize what can be done in connection with the examination of a joint of this magnitude.

Dr. Morrison didn't speak, of course, by reason of the lack of time, concerning his observations of the changes going on in the epiphysis for the head of the femur, with especial reference to osteochondritis (Perthes', Legg's or Calvé's disease). It is in this condition particularly that roentgen rays have served a definite purpose. We have found conditions unexpectedly perhaps, through our modern use of roentgen rays which, when the case was brought to us, was definitely believed to be tuberculosis because, as you know, all hip disease was known as "tuberculosis." Within the last year I know of three or four cases coming under my observation where Calvé's, Legg's or Perthes' disease was easily demonstrated, whereas clinically it was believed to be tuberculosis of the hip. As you know, the treatment differs so materially that you can imagine the importance of diagnosis.

Another point that Dr. Morrison has brought out is the question of the development of a tendency to congenital dislocation by reason of the changes in the marginal rim of the acetabulum. That is a very important clinical point, it seems to me.

I will close merely by saying that I feel we should pay more attention to the technique of the examination of this joint. In the examinathe of the alimentary tract it is necessary for us to turn our patient to portray various angles or planes. We do it constantly, both in roentgenograms and of course in fluoroscopy. But in the hip where fluoroscopy can't well be used, let us reveal the hip in various angles, always maintaining, of course, our impression of the standard position which we are all familiar with. Professor Köhler has shown, in a series of roentgenograms in the English translation of his book the same hip, at different angles, and I am sometimes in the habit now of using the old 23° sinus board which all are familiar with, pushed under the buttocks on the affected side, with a vertical projection from the anode, to show more of the acetabular cup or rim than I could show ordinarily. I have done this for many years in the examination of the glenoid cavity. You get a great deal of information by projecting the rays from different angles and studying them all together. You mustn't forget "Hickey's position" or lose sight of it, for it has great diagnostic possibilities.

Dr. Ralph Bromer, Philadelphia. I would like to ask Dr. Morrison if he has any explanation for the much more frequent occurrence of congenital dislocations of the hip in the female than in the male. Secondly, I believe Jansen, some years ago, put forth the theory that Legg's disease or osteochondritis of the hip joint occurred in children who showed a congenital acetabular defect. Because of this defect a change occurs in the lines of stress and strain in the head and neck of the femur, as well as in the acetabulum, and ultimately osteochondritis develops. I would like to ask Dr. Morrison if he has noticed congenital defects in cases of Legg's disease.

Dr. Albertus Cotton, Baltimore, Md. I consider Dr. Morrison's paper a very valuable one. I am sure that many of us have made mistakes in diagnosis owing to the fact that we

have overlooked the acetabular epiphysis of which Dr. Bishop writes. In adult cases where the epiphysis has failed to unite with the acetabulum and remains as a separate bone, the condition might easily be mistaken for fracture of the acetabulum or early hypertrophic arthritis. Inquiry into the history of the case, careful physical examination and a knowledge of epiphyseal development will prevent a mistake in diagnosis.

The technique of roentgen examination of the hip joint has been discussed. In addition to the routine plain and stereoscopic films of the hip and pelvis it is well to bear in mind the lateral oblique position advocated by Dr. Hickey several years ago. This method has been of assistance to me in a number of cases where it was important to bring out the exact location of the pathology in the upper end of the femur. It is scarcely necessary in a discussion before this Society to mention the importance of the proper position for taking anteroposterior stereoscopic or plain films of the hips. However, I have known mistakes in diagnosis of impacted fracture of the neck of the femur to be made because the technician neglected to keep the legs parallel and the feet straight up, where the affected side had been allowed to drop over into external rotation. Films taken in this position cause a foreshortening of the neck of the femur giving an appearance resembling impacted fracture of the neck of the femur.

Dr. Morrison (closing). These cases were all studied stereoscopically and many were asked to return and we took plates of them in various positions to bring out the situation more distinctly. In regard to Dr. O'Brien's question as to the relation of genetic centers and epiphyses, I shall define what I mean. As bone develops in any part little bony spicules will be noticed which invade the cartilage. These come together heterogeneously and fuse. The cartilage between them doesn't remain any length of time as the process is a rapidly progressive one. This is what I speak of as an ossification center. For example: when the ilium begins to develop it starts from a bony center and from this point it spreads out progressively producing more and more bone at the expense of the neighboring cartilage. A genetic center is that portion of bone at the end of the diaphysis which allows for further elongation and development in size. It is the growing center of an epiphysis. It is this center that I referred to when I was describing the accessory rim epiphysis of the acetabulum. A genetic center may be absent and then we note the deficiency of certain bony elements. This is frequently observed in the fibula, tibia and femur. An epiphysis is the growing portion of bone beyond the diaphysis separated from it by the conjugal cartilage. It is this connecting cartilage that remains intact while the epiphysis is reaching its primary growth and then ossification and calcification completely fuse it to the diaphysis. Genetic centers are the bone propagating areas within an epiphysis.

In a study of an epiphysis any of the transitional forms may be found. In many congenital cases the deformity is due to a complete absence of genetic centers with the resulting loss of bony elements. In this study this leads to the formation of a shallow acetabulum which may extend up as far as the posterior portion of the ilium or, as a second degree dislocation, appears even with the anterior inferior iliac spine. Pressure changes complete the picture of the deformity but are of secondary importance. When a genetic center is present without its epiphysis and conjugal cartilage the joint can be restored surgically to resemble the hip of the average ten year old.

Dr. Brown emphasized an extremely important point. It is most essential to use different positions and angles in taking plates of the hip if a complete picture of its condition is desired. I didn't go into particular diseases of the hip because the subject is so broad.

Dr. Bromer asks if there is any reason why the congenital hip should occur more frequently in the female than in the male. I don't really know; but I shall look up the proportion between the two. I know that Jansen speaks of it as having two factors. One is the fact that man's head is so much larger in proportion to that of the animals that it acts as a fulcrum for the legs to dislocate when pressure is brought to bear in utero. The other factor deals with the sexual difference in occurrence which he ascribes to the wide pelvis of the female. I cannot endorse his theories. Any explanation, I believe, must be built about genetic failures which may be further influenced by the general well being of the individual or some endocrine disturbance.

Vol. XXVIII, No. 4

A METHOD OF TREATING CARCINOMA OF THE VULVA*

By MILTON FRIEDMAN, M.D.
NEWARK, NEW JERSEY

THE great majority of epitheliomata of the vulva are epidermoid carcinomata of the squamous cell type, and are usually of a low grade of malignancy (Grades I and II). This is the usual type of cancer that grows in response to a long-standing irritation, which at first has induced benign hyperkeratotic changes in the skin, followed later by involution to malignancy. The resultant growth in this particular location, once it becomes malignant, loses the essential feature of indolence that characterizes Grades I and II epitheliomata elsewhere, and develops a tenacity for living and growing that few therapeutically destructive assaults can dispel.

Therefore, in planning the treatment of an epithelioma of the vulva, we find ourselves confronted with a slow-growing, malignant tumor, which appears to be fairly well localized in the early stages, thereby seeming favorable for radical surgery. For various known and unknown reasons, including the peculiar perniciousness of neoplasms of the vulva, the five-year cures are disappointingly small, averaging less than 10 per cent.[1] Therefore, we turn to radium and roentgen rays for assistance. For these same reasons, we find that the aid offered by radium and roentgen rays is small, but nevertheless it is a supplementary advantage.

The problem of treating carcinoma of the vulva is threefold: (1) destruction of the local lesion, which is simple; (2) destruction of residual cancer cells in the tumor-bearing area, which is more difficult, and (3) eradication of metastatic inguinal nodes, which is very difficult.

The local lesion may be treated by the interstitial insertion of gold radon seeds or by electrosurgical removal. Interstitial irradiation is not preëminently desirable because (1) the tumor is very radioresistant and requires intensive doses of radium to destroy it—10–14 S.E.D. in the tumor itself; (2) the secondary infection and inflammation of the ulcer during the radium reaction, which seems to be more severe in this area than elsewhere in the body, further enhances the probability of metastasis; (3) the intense dose required would delay healing, if not completely retard it, or would leave a painful radium scar which would have to be excised later; (4) this local irradiation would prevent the administration of subsequent external irradiation to the tumor-bearing area. This tumor-bearing area, which is a locus for recurrences, includes the vestibule of the vagina, the labia majora and minora, lateral borders of the perineum, the site of the tumor itself, the clitoris, and the lateral border of the mons in the inguinal fold.

It is preferable, therefore, first to remove the malignant mass with the electrosurgical knife. Only the tumor and the invaded tissues close to the tumor should be excised. A wide excision is undesirable because (1) the postoperative irradiation is less effective when bombarding a surgically injured area which contains potent implants of cancer cells than when attacking an undisturbed area; (2) the possibility of a large irradiated wound breaking down is greater than that of a small one; (3) the time required for healing of an extensive area which has been irradiated is additionally prolonged; (4) the proclivity of the knife for implanting the malignant cells in a larger subcutaneous garden is augmented.

After the tumor is removed, the edges of the wound are sewed together. This suture line usually breaks down in varying degrees during the subsequent radium reaction.

* From the Radiation Therapy Departments, Bellevue Hospital and Sydenham Hospitals, New York City; and Beth Israel Hospital, Newark, New Jersey.

RATIONALE OF RADIUM TREATMENT

We now have an extensive area, in any single portion of which may lurk a cancer cell. This is the tumor-bearing area described above. It is necessary to expose this area to the greatest possible quantity of gamma rays necessary for the complete destruction of the residual cancer cells, and yet compatible with the continued good health of the normal tissues.

The only absolutely effective method of administering radium is by means of the widespread distribution of gold radon seeds homogeneously throughout the tumor-bearing area so as to provide a dose of 10–14 S.E.D. to the tissues. Unfortunately, this is impractical because (1) the tremendous dose would cause an intensely painful reaction, and radium burn with ulceration, which would debilitate and devitalize the patient because of the prolonged torture, and probably cause death; (2) the open wound would invite secondary infection, especially since urine and feces are constant threatening contaminants to the dressings; (3) it would be impossible to implant radon seeds throughout this area evenly because of the well-known inability to maintain the theoretically homogeneous dispersion of the seeds as outlined during their insertion (Fig. 3, *1*), and as a result, part of this tumor-bearing area would be sufficiently irradiated so as to destroy all remaining malignant cells, and other parts would not, thereby invalidating the successful result attained elsewhere; (4) furthermore, a large quantity of radon would be very costly.

Since interstitial irradiation is inexpedient, we must resort to an effective method of external irradiation. The latter could not provide a sufficient dose to sterilize the area thoroughly, but would repress the tendency for recurrences, and retard the rate of growth of the recurrent nodules, converting them into sporadic, slow-growing, localized lesions, which could then be attacked individually by the implantation of measured amounts of radon. The more radiosensitive epitheliomata of the vulva (Grades III and IV) might be completely and effectively controlled by this external irradiation alone.

The depth of the tumor-bearing area averages 1.5 cm. and seldom exceeds 2 cm. If we apply heavily filtered radium (3–4 mm. lead filtration or its equivalent) on a wax mold 1 cm. from the skin, there would be a depreciation in the intensity of the radiation to about 80 per cent of the skin intensity in the first centimeter of depth, and 50 per cent in the second centimeter. (These latter two figures are estimated, not calibrated.) By this means, if the treatment were carefully planned so as to provide an optimum time interval, we might deliver to the skin in a period of two weeks 3–4 S.E.D. of a very energetic type of radiation.

The ensuing radium reaction is transient and not very painful. The highly penetrating, short wave gamma rays employed are more ably tolerated by the normal tissues, and cause greater destruction to the neoplastic cells than an equivalent dose of poorly filtered radon emitting the softer, more caustic, less selective, and less penetrating gamma rays.

The inguinal nodes are best treated by radiation alone. Only 60 per cent or less of palpable nodes in carcinoma of the vulva are malignant. Secondary infection of the original lesion with adenitis accounts for the enlargement of the other 40 per cent of the nodes.

Coexisting carcinoma of the opposite side of the vulva, whether it be metastatic or transplanted, adopts the characteristic of lesser virulence in that it remains localized for a longer period of time, does not metastasize of itself until late, and responds well to radiation. The radiation directed at the primary lesion is usually sufficient to control this transplant or contact epithelioma.

TECHNIQUE OF RADIUM APPLICATION

Radium Mold. External irradiation of the tumor-bearing area postoperatively is accomplished by holding the radium in

position with the aid of a wax mold (Fig. 1). This irradiation may be carried out at the conclusion of the operation, or in four days, when primary union has commenced

Fig. 1. Wax mold showing radium in place.

and the sutures have not yet been removed. The patient should be placed in lithotomy position with the thighs at right angles to one another, and with each knee supported at right angles with a pillow.

Cut out a large triangular plaque of warmed and softened Columbia molding wax impregnated with sawdust and 1 cm. thick. Truncate the apex slightly; bend the apical third of the triangle at right angles to the rest and mold that into a tube. Insert the tube into the vagina, placing the rest of the mold against the perineum. Distend the tubular portion of the mold as far as possible in the vagina with the fingers. Compress the perineal part gradually so as to conform to the contours of the area. Bend the lateral wings of the perineal part of the mold to fit against the thighs so that it holds them apart. Two strings tied to the upper part of the mold and fastened to an abdominal binder, together with a T-binder, hold the mold securely in place. If the mold is well lubricated with vaseline, it is a simple procedure for the nurse to remove it and replace it securely, thereby permitting urination and defecation. There is practically no distress to the patient during this treatment.

Radium. The radium tubes are melted into the mold in the usual fashion with a melting iron. The radium itself should be filtered very heavily with 3–4 mm. of lead or its equivalent, and employed in units of 5–25 mg. of radium in each tube. If the tubes or needles have insufficient filtration, they can easily be wrapped in lead sheeting of measured thickness. This heavy filtration is desirable because it absorbs the less penetrating rays, and permits the egress of very highly penetrating gamma rays of short wave length. In the opinion of one school of thought, these rays exhibit a greater selectivity for the malignant cells, produce less injury to the normal cells in comparison with an equivalent dose of softer, less penetrating rays of longer wave length, and of most importance, permit the administration of $2\frac{1}{2}$ to 3 S.E.D., as measured by physical means and yet produce only one biological or clinical S.E.D.[2] Furthermore, if only part of the total dose is given every day, or every other day for two weeks, the skin and normal tissues will tolerate an even greater quantity of radiation because of the daily depreciation of the radiation effect.[3]

The wax mold is 1 cm. thick. The wall thickness of the radium tube is 3–4 mm., making a total radium-skin distance of 13–14 mm. Usually two tubes are placed in the vagina (see Fig. 3, *3*); three, if the growth has involved the clitoris. The rest are distributed on the mold to surmount the labia and perineum up to the inguinal fold. From 8 to 12 tubes may be employed. The smaller tubes or needles of 5 or 10 mg. are most preferable because they permit the administration of the dose over a longer period of time.

Dose. Certain dosage factors are pertinent: (1) One skin erythema dose produced by a radium tube 14 mm. long, filtered with

Fig. 2. Radium mold fitted in place. The tendency of the vagina to extrude the vaginal portion of the mold is counteracted by the application of a T-binder. The knees are supported in a flexed position with pillows.

2 mm. lead, and placed 1 cm. from the skin is equal to 236 mg-hr. of radium.[4] (2) The S.E.D. with 3 mm. lead filtration is 255 mg-hr. (3) The S.E.D. with 4 mm. lead filtration is 400 mg-hr.

To apply the above figures to the calculation of the dose in a specific case, let us assume a hypothetical lesion as illustrated in Figure 2. There are eight tubes on the wax mold which is 1 cm. thick. These tubes are filtered with 4 mm. lead or its equivalent, 2 mm. platinum. The biological S.E.D. for each tube is 400 mg-hr., and for the whole mold is 3,200 mg-hr. However, this is only the first constant calculated, and must be modified, before the final dose is estimated, by many variable factors, the knowledge of whose value comes to the operator only with experience.

The factors necessitating a slight decrease in the originally calculated dose are:

1. Close proximity of two or more tubes which mutually intensify the quantity of radiation delivered to the underlying skin.

2. Cross-firing of tubes inside and outside the vagina (see Fig. 3, *3*).

The factors commanding a corresponding increase in the dose are:

1. The additional wall thickness of the radium tube (which is particularly increased when lead filtration is employed instead of platinum) increases the radium-skin distance, and consequently diminishes the intensity of the radiation that reaches the skin.

2. The biological S.E.D. is based on the "threshold erythema," whereas the skin can actually tolerate a 50 per cent greater dose, especially when heavy filtration is employed.

3. Assuming that the treatment will be given in one continuous dose lasting for one to three days, it is well to remember that if smaller quantities of radium are employed in units of 5 or 10 mg. tubes, and the radium is of necessity left in place for a longer period of time, the tissues can withstand a greater quantity of radiant energy without greater damage. This is due to the fact that every twenty-four hours, there is a loss of approximately 20 per cent

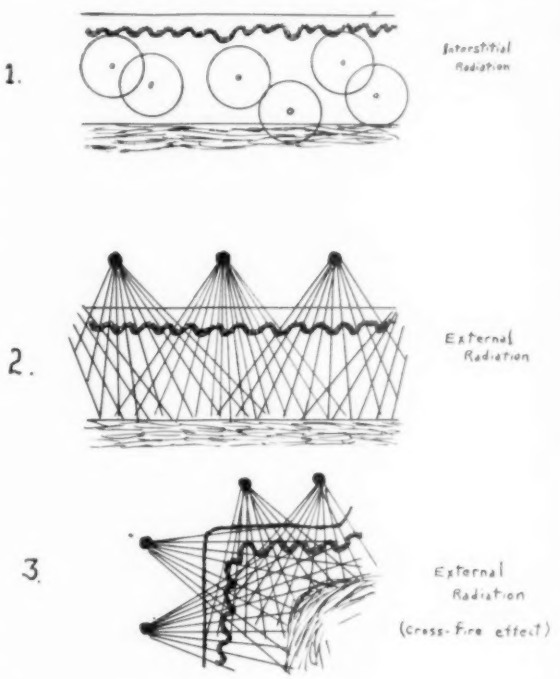


Fig. 3. *1.* Diagram showing spherical areas of tissue in the tumor-bearing area thoroughly irradiated by means of implanted gold radon seeds. Unequal distribution, which is unavoidable, permits scattered areas of insufficient irradiation, and subsequent recurrences. *2.* Beneficial effect of cross-firing of radiation from coadjacent radium tubes on a wax mold. *3.* Cross-firing effect of radium from inside the vagina and on the perineum upon that portion of the vulva which is most frequently the site of carcinoma.

of the absorbed energy in the skin. On the succeeding day, that additional amount can be administered without causing further damage to the skin and underlying structures. This is the "saturation loss effect."[3]

4. Should the treatment be administered in partial daily doses over a period of two weeks, then almost double the original quantity of radiation can be administered.

Since most of the above-mentioned variables have not yet been converted to constants suitable for practical use by experimental laboratory and clinical evidence, we must rely on the personal judgment of the radiation therapist in applying the facts to the individual case.

In conclusion, therefore, if the treatment is to be administered in one sitting, one can safely allow 600–700 mg-hr. for each tube. If the treatment is to be administered over a period of two weeks in partial daily doses, one can safely allow 1,100–1,200 mg-hr. for each tube. If a full treatment is to be given, and then repeated in two weeks, one can allow 500 mg-hr. for each tube.

TREATMENT OF INGUINAL NODES

The eventual success of the treatment depends more on the eradication of the stubborn metastatic invasion of the inguinal nodes than on the treatment of the local lesion. The combination of external and interstitial irradiation in treating these glands is more successful than surgery alone, or surgery plus irradiation.

External irradiation is accomplished with high voltage roentgen therapy or radium pack. When employing roentgen therapy, the voltage, filtration and distance are not as important as the total quantity of radiation administered, because the glands are superficial and do not require deeply penetrating rays to reach them. The utilization of the saturation technique of daily divided doses[3] permits the administration of 250 per cent S.E.D. in two weeks with no damage to the normal tissues. If a radium pack is used, the skin-target distance need not exceed 4 cm., and a small quantity of radium (100–200 mg.) may be used in order to provide a dose of 10,000–12,000 mg-hr. over each area.

Following this, gold radon seeds should be implanted in the nodes to bring the dose in the tissues up to 10–12 S.E.D., according to the method so admirably outlined by Martin and Quimby.[5] The quantity of radon should be reduced in those areas close to the femoral vessels for fear of thrombosis or rupture.

SUMMARY

A method for treating carcinoma of the vulva by the judicious utilization of surgical endothermy, radium, and roentgen rays has been outlined.

REFERENCES

1. RENTSCHLER, C. B. Primary epithelioma of the vulva; analysis of 71 cases. *Ann. Surg.*, 1929, *89*, 709–730.
2. QUIMBY, EDITH H. Comparison of different metallic filters used in radium therapy. AM. J. ROENTGENOL. & RAD. THERAPY, 1925, *13*, 330–342.
3. PFAHLER, G. E., and WIDMANN, B. P. Further observations on the use of the saturation method of radiation therapy in deep-seated malignant disease, with some statistics. *Radiology*. 1928, *11*, 181–190.
4. WIDMANN, B. P. Physics of Radiology. Paul B. Hoeber, New York City, 1931.
5. MARTIN, H. E., and QUIMBY, EDITH H. Calculations of tissue dosage in radiation therapy; preliminary report. AM. J. ROENTGENOL. & RAD. THERAPY, 1930, *23*, 173–196.

UNFILTERED ROENTGEN RAYS FOR SUPERFICIAL CANCERS OF WIDE AND DEEP INVOLVEMENT*

By BERNARD P. WIDMANN, M.D.
PHILADELPHIA, PENNSYLVANIA

ROENTGEN rays, radium, electrocoagulation, or various combinations of these agents, are universally accepted as of curative value in the treatment of superficial cancers. In a series of 220 traced skin cancers at the Philadelphia General Hospital, the superficial lesions are symptom-free two years or more in 87 per cent of 156 cases, as compared to 57 per cent in the deep, infiltrating superficial lesions (64 cases). The latter group were late cases, complicated by infections and generally with muscle, bone or cartilage involvement.

These results can be obtained with a wide range of techniques in the hands of experienced radiologists. The best results are achieved with technical procedures that suit the particular case. It is obvious that no criteria for the treatment of these lesions will hold for all cases, because feeble and elderly persons are not always suitable for the general anesthesia necessary for an extensive electrosurgical destruction; hospitalization, office or clinic visits with inconvenient and distant residence, as well as other physical and economical considerations must be weighed in determining the choice of treatment.

Any procedure that will cause slough over a wide area will necessarily involve considerable toxic absorption and weakness sufficient to prolong the period of hospitalization. Heroic measures in advanced cancer—even though it is an extensive so-called skin or superficial cancer—are always likely to precipitate a patient into a condition of misery and suffering with grave possibilities of recurrence. Unless the first treatment is thorough and adequate, the likelihood of recurrence or failure is great.

During the past two years many extensive superficial cancers have been encountered in individuals not physically fit to stand the combined irradiation and electrosurgical procedures ordinarily employed for smaller lesions. In many instances the involvements were obviously hopeless—from wide destructive infiltrations of bone and cartilage.

Conservative procedures with filtered roentgen rays and radium (both surface and interstitial) of varying qualities have been successful in many instances.

There is still much demand for a simple and accurate technique that might be more nearly ideal. Unfiltered roentgen rays (127 kv., 0.45 Å) have been extraordinarily satisfactory and successful in the treatment of small lesions of about 1 to 2 cm. in diameter. This size of lesion has shown good regression and healing with as much as 5 to 10 S.E.D., depending on the thickness of the growth (usually given in divided doses).

The administration of intensities of unfiltered roentgen rays in excess of 2 to 3 S.E.D. to lesions greater than 2 cm., i.e., lesions 5, 8 and 10 cm. in diameter, necessarily involves great risk of effecting a radiation necrosis.

This study, therefore, is an attempt to evaluate unfiltered roentgen rays for the treatment of large inoperable skin cancers—5, 8 and 10 cm. in diameter—and the clinical results obtained with doses of 3, 5, 10 and 20 S.E.D. Various criteria with different intensities are submitted, individually and collectively. Specimen cases representing various anatomic sites are discussed and analyzed, the limitations of various technique defined, and recommendations for improved end-results submitted.

* From the Radiological Department of the Philadelphia General Hospital, Philadelphia. Read at the Thirty-second Annual Meeting, American Roentgen Ray Society, Atlantic City, N. J., Sept. 22–25, 1931.

DOSAGE CONSIDERATIONS

A dose of unfiltered roentgens rays (S.E.D.) is generally accepted to imply a threshold erythema which should be obtained in about 80 per cent of the cases.

The reaction appears in about ten to fourteen days and subsides in about ten days. Depilation occurs in about fourteen to eighteen days. A mild redness or blanching with subsequent scaling represents the minimum erythema dose as contrasted with a deep redness of the skin followed by blistering and desquamation compatible with the maximum skin tolerance to radiation. This latter reaction will appear in about eight to ten days and disappears in about three weeks, terminating in a bronzing which frequently persists for several months.

Blanching and redness of the skin depend so largely on the element of personal equation that accurate physical measurement controls are now indispensable.

Unfiltered roentgen rays (100 to 130 kv.) may show energy differences that vary as much as 50 to 100 per cent according to the type of mechanical rectification. Fluctuating currents, faulty meters, the variable glass wall thickness of the tubes, as well as the extent of metallic coating on the walls from pitting of targets due to hard and prolonged use, are factors that seriously alter the output of radiation. Ionization measurements or the "half-layer value" tests must be carried out routinely for all equipments. The output of unfiltered roentgen rays is not constant for different tubes even with the same potential and the same load.

The interpretation of an erythema has been so variable that the confusion and difficulties are readily appreciated by the wide range of roentgens set forth by various authors to indicate the erythema value or the skin tolerance of a single roentgen treatment that can be safely given on a normal skin area.

The range for the minimum and the maximum dose is variously quoted from 250 to 2,500 r for fields less than 1 to 2 cm. in diameter; for larger fields, 10×15 cm., and 20×20 cm., the latitude ranges between 250 and 1,200 r (for filtered and unfiltered roentgen rays alike).[1,2,4,6,8,9,10] These differences represent the minimum and maximum skin reactions variously reported in the literature. Obviously, criteria for erythema estimations must be controlled by physical measurements.

It has been generally conceded, experimentally and clinically, that the biological effects of long and short wave length rays are similar, the difference being one of absorption and penetration. Neoplasms, involving depths greater than 3 cm., and especially 5 to 10 cm., can therefore be affected with short wave length rays to a much greater and more accurate degree and with less risk of skin damage than with long wave length roentgen rays.

Our own experience with different qualities of roentgen and radium rays for cancers of the lip is convincing evidence of similar biological effects because the end-results were equally good for early operable cases of approximately equal involvement.

Wood[12] concludes from many animal tumor experiments that there is no difference in the lethal effect of long and short wave length radiation.

Failla and Henshaw,[3] in an exhaustive study of the biological effectiveness of radiations, found that no matter how the radiation is measured, there is a distinct difference in the effectiveness of roentgen and gamma rays, for some biological effects. They conclude from experimental results that the quantity of radiation required to produce a certain degree of effect in a given tissue depends on the quality of the radiation. They noted that the greater relative effectiveness of gamma rays is not the same for all tissues examined. Gamma rays were found to be 2.9 times as effective as roentgen rays in reducing the growth of the shoots of the wheat seedlings, but they were 4.1 times as effective in killing *Drosophila* eggs. In producing skin erythema this greater effectiveness is only 1.2 times that of roentgen rays.

Epilation is a decidedly measurable biological effect for long wave length radiation, but is not always coincident with the time of appearance of the erythema. The hair of the scalp will epilate with 70 per cent and very often with 60 per cent of a skin erythema dose; the bearded areas of face and neck require 90 per cent S.E.D.; the pubic and axillary regions usually require 100 per cent S.E.D. for epilation. These factors represent our own experience and are at variance with some observations of others.

Epilation with short wave length radiation necessitates a greater dose in terms of roentgens. During the past three years we repeatedly noted intense erythemas from short wave length radiation and frequently epilation occurred one to two weeks after onset of erythema and very frequently no epilation was even observed.

Our own clinical experience, supported by ionization and "half-value layer" estimations, indicates that the skin is decidedly less tolerant for long than for short wave length roentgen rays (areas 5, 10, 15 and 20 cm. in diameter). The effect of back-scattering is greater for hard than for soft rays but there is no agreement on the nature and extent of this effect. No measurable back-scattering has been noted for very small areas of 1 and 2 cm. (as measured in air).

Glasser and Meyer[4,7] found that the erythema dose was markedly dependent on the wave length and that more than double the amount of hard rays (1,300 r) was necessary to produce the effect of soft rays (500 r). Pohle and Barnes[8] found a slightly greater intensity of redness with unfiltered roentgen rays (130 kv.) as compared with 0.5 mm. of copper filter (200 kv.), employing 600 r for both treatments. They did another series of control exposures at 130 kv. (unfiltered, 1, 2, 3, 4 mm. Al., 0.25 mm., Cu plus 1 mm. Al), with increasing energy. Theoretically the values used should represent threshold values for the erythema, but no uniform reaction was observed.

Marie[5] measured the intensity of radium needles arranged in a suitable pattern over a large area and observed a great increase in the depth dose. He found no evidence that the gamma rays of radium act upon the individual cells in any different way from that of the gamma rays given off by the roentgen-ray tube. The destruction of the individual cell, which is probably due to the electron energy released in the cell, is accomplished, it is most logical to believe, by the slow-moving secondary and tertiary electrons, rather than by the high speed ones. Marie believes it is theoretically improbable that there will be any difference between the effects of equal quantities of radiation of any wave length.

Chamberlain and Newell[1] found no difference in the epilation or erythema produced by equal doses (in r-units) of hard (λ-eff. 0.17 Å) and soft (λ-eff. 0.45Å) roentgen rays (doses varied in 20 per cent steps from 1,600 R down to 215 R).

TECHNIQUE

At the Philadelphia General Hospital, Weatherwax established the maximum safe erythema dose for unfiltered roentgen rays (127 kv.) as 500 r, and 800 r for 200 kv. and 0.5 mm. of copper.

Weatherwax and Robb[10] have recently made further studies which conform to the findings of Glasser and Meyer that the erythema values depend on the quality of roentgen rays used. (All measurements made in air.) For example:

0 filter	127 kv.	= 500 r
4 mm. Al	127 kv.	= 600 r
6 mm. Al	127 kv.	= 650 r
0.5 mm. Cu	200 kv.	= 800 r

Weatherwax and Robb found that 1.5 cm. of water will absorb as much radiation as 0.5 mm. of aluminum (127 kv.); that 2 cm. of water will absorb as much radiation as 1 mm. of aluminum, and that 3.2 cm. of water will absorb as much as 2 mm. of aluminum.

In actual clinical work, therefore, the first centimeter of tissue will absorb about 22 per cent of unfiltered roentgen rays (127 kv.); 2 cm. of tissue will absorb 44 per cent;

3 cm. of tissue will absorb about 60 per cent, and 4 cm. about 67 per cent.

In practical clinical therapy it was felt that ulcerating neoplasms presenting a depth of 0.5, 1, 1.5 and 2 cm. and even more would be ideal for massive intensities of long wave length radiations. The sloughs and fungations should readily filter out the caustic qualities and afford a tissue filtration that would give depth doses sufficiently adequate to destroy cancer without serious destructive effects on the deeper lying normal tissues.

On the basis of these observations, an attempt was made to lay down some mathematical procedure that would admit of a greater intensity of unfiltered roentgen rays without producing a dangerous radiation necrosis for fields of 5, 10 and 15 cm. diameter.

Small areas of 1 and 2 cm. diameter have been given 5, 10 and 15 S.E.D. (2,500 to 7,500 r) with excellent clinical and cosmetic results. The thickness of these lesions ranged from 2 to 5 mm. All lesions showed infiltrating margins. An excess of 3 S.E.D. was never given unless there was obvious infiltration.

Large ulcerating lesions 5, 10 and 15 cm. in diameter, involving exuberant granulations with fungations 1 to 5 cm. in depth, were given similar doses of unfiltered roentgen rays (127 kv.) of 5, 10 and 15 S.E.D. (2,500 to 7,500 r), and usually given in doses of 3 to 5 S.E.D. once a week.

In several instances 20 S.E.D. once a week (10,000 r) in divided doses were given but these cases presented extraordinarily large fungating neoplasms.

The following factors were employed as standardized by Weatherwax.*

1. Unfiltered roentgen rays: 127 kv. (crest value with 12.5 cm. spheres); 5 ma.; mechanical rectification, effective wave length 0.42 Å; erythema dose equals 500 r.

2. Filtered roentgen rays: 200 kv. (crest value with 12.5 cm. spheres); 30 ma.; water-cooled tube; mechanical rectification; effective wave length 0.16 Å; erythema dose equals 800 r.

3. Gamma radium rays: filter 0.5 mm. silver plus 3 mm. lead (in contact gives intense erythema with 350 mc. at distance of 5 mm. This combined metal density is approximately 2.5 times greater than the density of 2 mm. of brass).

The areas treated were 3 to 15 cm. in diameter. Epilation and erythema intensities are not recorded for these cases because the radiation was sharply localized to fungating ulcerations.

An experience with doses of unfiltered roentgen rays involved:

2 S.E.D. (1000 r) weekly for 3 doses (3000 r)
3 S.E.D. (1500 r) weekly for 2 doses (3000 r)
3 S.E.D. (1500 r) weekly for 3 doses (4500 r)
5 S.E.D. (2500 r) weekly for 1 dose (2500 r)
5 S.E.D. (2500 r) weekly for 2 doses (5000 r)
5 S.E.D. (2500 r) weekly for 3 doses (7500 r)
5 S.E.D. (2500 r) weekly for 4 doses (10000 r)

Contact gamma radium applications were combined with the above factors in selected cases, particularly in infiltrating lesions. The radium intensities were usually equal to the total intensites of applied unfiltered roentgen rays but the irradiated area with radium also included the surrounding normal skin (about 1 to 3 cm.). Only the ulcerated areas received these combined qualities of radiation.

When high voltage roentgen rays were employed with the above factors the technical procedures of limiting the total skin dose to 150 per cent S.E.D. on basis of saturation as set forth in another paper by me[11] were adhered to.

At no time was the unfiltered roentgen radiation permitted to extend to the adjacent normal skin margin beyond 2 to 4 mm.

The approximate thickness of the exuberant fungations or granulations and the square surfaces of the lesions were determined in millimeters or centimeters (estimated and photographed—not always actually measured).

* All ionization measurements made at Philadelphia General Hospital by Weatherwax and Robb are records of air intensities.

MATERIAL

During the past three years 104 cases of superficial cancer have been treated. Forty-six cases with wide and deep involvement were treated with unfiltered roentgen rays. (Rodent ulcer, 9 cases; lip, 14 cases; breast, 6 cases; other skin areas, particularly about the face, 17 cases.)

Twenty-two cases were treated with combinations of unfiltered and filtered roentgen rays, and gamma rays of radium. (Rodent ulcer, 4 cases; lip, 7 cases; breast, 2 cases; penis, 2 cases; other skin areas, especially about the face, 7 cases.)

There were 36 early cases studied from the standpoint of different techniques.

Lesions of similar anatomic sites, of like cellular characteristics and with involvements of apparently equal extent showed a wide range of responses. These differences were especially marked for the squamous cell types of cancer.

Any lesion that failed from previous treatment with roentgen rays or radium or electrodesiccation, or a recurrent lesion from such previous treatment, was not amenable to additional effects of unfiltered roentgen rays. In exceptional instances, regressions of resistant or recurrent lesions were noted when the quality of additional radiation was changed—particularly toward the short wave length side.

The recurrent and resistant lesions generally were more successfully managed by complete electrocoagulation and then supplemented with short wave length roentgen rays or radium packs. Complete eradication by surgical or electrosurgical procedures is almost always necessary for the resistant or recurrent lesions because of the extensive fibrosis, hyalinization and diminished circulation from the diffuse obliteration of the finer capillary blood vessels—the endarteritis which goes hand in hand with prolonged or heavy irradiation making a bed of tissue most unsuitable for any good effects from further irradiation.

Superficial lesions, 1 and 2 cm. and also 4 to 6 cm. in diameter, with granulations of 1 to 2 mm. thickness, required about 3 S.E.D. unfiltered roentgen rays and showed excellent results with soft, smooth scars and without telangiectasis. Two S.E.D. (1,000 r) once a week for two to three treatments (2,000 to 3,000 r) offered equally good results. Any dosage in excess of this frequently resulted in delayed healing, and the risk of thick telangiectatic scars is always imminent.

There is always the obvious element of uncertainty in estimating an exact dose and defining a clinical cure. Residual thickenings and local reactions to inflammation are sometimes aggravated by radiation reactions and confound decisions on the possible necessity of additional radiation. The anxiety as to whether sufficient treatment had been estimated is further accentuated by the slow regressions usually extending over a period of four to eight weeks.

For these reasons all small superficial cancers (except lesions of the eyelid) are routinely treated with complete removal by electrodesiccation. Biopsy, complete destruction and postoperative roentgen treatment are done at the same sitting. The procedure is as follows:

Destruction is done under novocaine anesthesia (1 per cent) by electrodesiccation after biopsy. The charred surface over the base of the wound is thoroughly curretted to avoid leaving intact any vestige of soft bleeding granulation tissue, which clinically should always be held as evidence of remaining disease. A sharply localized dose of roentgen rays is applied (2 S.E.D., 2 mm. aluminum, 127 kv., for squamous cell lesions to be repeated in one week; 1 to 2 S.E.D., no filter, 127 kv., for basal cell lesions according to the extent of infiltration).

Usually no further treatment is necessary. The experienced operator is at once satisfied that the disease has been completely eradicated (for basal cell cancers) and the additional irradiation safeguards him against the possibility of deeper lying cells enmeshed in firm and apparently healthy tissue (usually squamous cell lesions). The additional irradiation also

serves to promote a soft smooth scar, particularly if there is any tendency to keloid formation.

For squamous cell lesions, filtered radiation of necessity must supplement electrodesiccation, because the microscopic pattern shows neoplastic cells dovetailing into the surrounding normal structure, so that electrodesiccation alone would obviously involve the unnecessary destruction of much normal tissue. Even then the disease could not always be entirely destroyed. Frequently the metastatic lymph nodes may be involved without clinical evidence of such extension.

Superficial cancers of wide and deep involvement and clearly inoperable are not usually ideal for a thorough and adequate electrosurgical removal. Recurrences are frequent even after painstaking and extensive destructive procedures.

In order to ascertain whether a more conservative and yet efficient radiation technique might be adequate for these cases, an intensive and systematic clinical study was made on the effects of large doses of unfiltered roentgen rays. Extensive rodent ulcers with bone and cartilage involvement showed startling results within a short period of time (about four to six weeks) with surprisingly large doses of unfiltered roentgen rays.

In some instances the doses exceeded the findings of Weatherwax on the absorption of radiation in the first, second and third centimeters of neoplastic tissue, and the unusual tolerance for the large doses outlined in the following case reports is not readily explainable. On the other hand, some very resistant and recurrent lesions did not show a commensurate tolerance or response with resulting wide breaking down of tissue and protracted healing.

The following cases are discussed in detail because they are substantially representative of the various types of involvements that have been subjected to large intensities of unfiltered roentgen rays.

A B

Fig. 1. Case 1. Rodent ulcer; complete clinical regression for two years. Death from uremia. Treatment: 12 s.e.d. (6,000 r) unfiltered roentgen rays (127 kv.) to five different areas with overlapping doses with a total radiation of 34 s.e.d. (17,000 r). A—March 3, 1929; B—June 11, 1930.

A B

Fig. 2. Case II. Rodent ulcer; clinical regression for six months. Death from pneumonia. Treatment: 18 S.E.D. (9,000 r) administered within three weeks on the basis of 6 S.E.D. once a week. A—May 18, 1929; B—October 1, 1929.

Case I (Fig. 1). A rodent ulcer showed fungations to the extent of 1.5 cm. thickness in the center of the ulcer and varying from 5 to 10 mm. about the margins of the ulcer. Unfiltered roentgen radiation was applied to five fields, cone 5 cm. diameter, directing the central beam from the center to the periphery, carefully protecting the normal skin with lead about 5 mm. beyond the visible margin of the disease. The margins of each field of radiation overlapped. The intensity of the overlapping radiation about the periphery of the ulcer was 12 S.E.D. (6,000 r), and in the center of the lesion 34 S.E.D. (17,000 r). Treatments were completed in one week. The foul, sloughing, bleeding mass regressed within six weeks. This man was sixty-seven years old, and physically was not fit for general anesthesia. Electrosurgical removal was contraindicated because of the wide involvement. There was no clinical evidence of recurrence within two years. Death was from uremia. There was no active ulceration and no local changes to indicate radiation necrosis. Skin margins were soft and smooth. Slight infection persisted about the margin of exposed dead bone (superior maxilla). The mucous membrane of nasal septa and remaining palate was of normal color and intact.

Case II (Fig. 2). Rodent ulcer of the nose. The thickness of the overlying granulations was about 6 mm. about the margins and 8 to 10 mm. in the center. In eleven days, 18 S.E.D. (9,000 r) unfiltered roentgen rays was given (3 treatments of 6 S.E.D. each at weekly intervals) with complete regression within six weeks. No recurrence and no radiation necrosis was noted within six months. Death was due to pneumonia.

Case III (Fig. 3). Rodent ulcer over parotid region was markedly infiltrating with a thickness of overlying granulation tissue about 3 to 4 mm. The infiltrating character of this growth was much greater than can be seen from the illustration. Unfiltered roentgen radiation was localized to this region—6 S.E.D. (3,000 r). Contact radium plaques were applied (0.5 mm. of silver and 1 mm. of lead) at the same time and within a period of six weeks a total of 12 S.E.D. (9,600 r) additional radium radiation was attained. (Total of the two qualities of long and short rays delivered was 18 S.E.D., or 12,600 r.) The radium filtration in this instance was 2 mm. of lead less than is now routinely employed for heavy doses in the infiltrating lesions. The thin granulating surface was totally inadequate to filter out the caustic effects of the large dose

A B

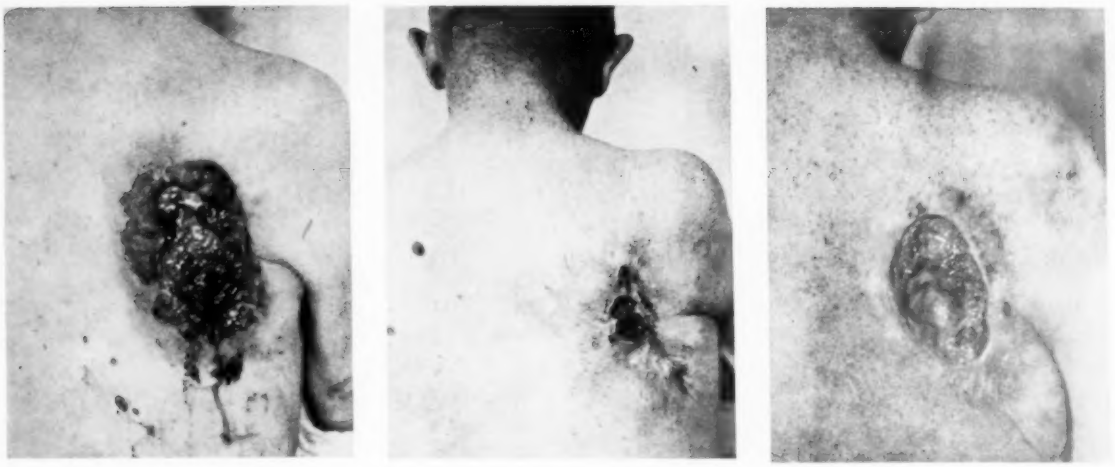
Fig. 3. Case III. Rodent ulcer; delayed healing about one year. Biopsy of margins and center of ulcer show no evidence of malignant disease at this time. Treatment: 18 S.E.D. (12,600 r) with combined unfiltered roentgen rays (127 kv.), 6 S.E.D. (3,000 r) and radium—12 S.E.D. (9,600 r), filter 0.5 mm. silver plus 1 mm. of lead. A—April 24, 1930; B—December 3, 1930.

administered. A typical radiation necrosis occurred which unnecessarily delayed healing about one year.

This experience was noted in two squamous cell lesions and led to a revision of technique—unfiltered roentgen rays limited to 2 S.E.D. at one sitting and repeated once or twice at weekly intervals according to the thickness of the surface growth (never to exceed 10 S.E.D.—5,000 r for 1 cm. thickness of tissue), and always supplemented with additional heavily filtered surface radiation—usually contact radium plaques (filter: 0.5 mm. of silver and 3 mm. of lead; total intensites of 2 to 3 S.E.D.—about 2,500 r). Adjacent glandular areas are always treated with high voltage roentgen rays.

Case IV (Fig. 4). This case illustrates the dangers and complexities that frequently arise in the treatment of recurrent lesions that previously received heavy irradiation. The specimen was microscopically a benign cystic epithelioma but as the lesion progressed, one month later another specimen showed a squamous cell carcinoma (two separate biopsies). Clinically a complete regression was obtained with 9,209 mc-hr. of interstitial gold seed (0.2 mm. gold) irradiation. Two years later nodular recurrences in the center of the scar grew progressively larger with infiltration and axillary and supraclavicular metastases. The metastatic nodes subsided with high voltage roentgen radiation distributed over the entire right chest. Unfiltered roentgen radiation was localized to two ulcers over the original site of the lesions, 2.5 and 5 cm. in diameter respectively—with a total of 5 S.E.D. (2,500 r) to each area. The breaking down of tissue was rapid and diffuse and eventually involved an area about 6×10 cm. Six months later a ridge-like border (squamous cell carcinoma) along the inner margin 1.5×6 cm. was given an additional 3 S.E.D. (1,500 r) with a resulting deep furrow from a gangrenous slough extending to the bone. Within a year healthy granulations appeared. The wound is not healed but is covered with a clean, solid, granulating base and healthy margins.

The long duration of the disease, the transformation of a benign cystic epi-



A B C

FIG. 4. Case IV. Squamous cell carcinoma engrafted on a benign cystic epithelioma. Complete regression for two years with gold seed implantations 9,209 mc-hr. Local recurrence was given 5 S.E.D. unfiltered roentgen rays with widespread breaking down of tissues. No local evidence of recurrence, but ulcer has not healed since treatment one year ago. A—November 23, 1927; B—March 25, 1929; C—November 1, 1929.

thelioma into a squamous cell type with axillary and supraclavicular metastases, and response of regional lymph node metastases to treatment with apparent clinical cure five years after onset of disease are significant and outstanding factors in this case. The diminished tolerance for unfiltered roentgen rays in this instance is probably accentuated by the diminished resistance of the tissues from the previous heavy interstitial radiation, and the very thin layer (2 to 3 mm.) of overlying granulation tissue.

The following cases illustrate various points of technical discriminations successfully employed in the treatment of advanced superficial lesions.

CASES V and VI. (Figs. 5 and 6). Deep infiltrating squamous cell cancers treated with two unfiltered doses of roentgen rays, 2 S.E.D. and 3 S.E.D. (1,000 and 1,500 r), with one week interval between treatments. Radium was applied during the same period of time. Capsules were arranged in plaques so that all surfaces of ulcerated areas, including a border of 2 cm. of the adjacent margin of healthy skin, were given intensities of 3.5 S.E.D. High voltage roentgen rays (200 kv., 0.5 mm. Cu) were administered to the adjacent lymphatic area of the neck and included the regions treated with unfiltered roentgen rays and radium (to saturation—150 per cent S.E.D.). The thickness of the neoplastic tissue was 1 cm. The total intensity of these combined qualities of radiation on and about 2 cm. beyond the margin of disease was 10 S.E.D. (6,000 r).

Both of these cases illustrate good cosmetic results. The radiation reaction involves considerable edema and desquamation, subsiding completely within six to eight weeks. The procedure is conservative and has been tolerated by even very advanced cases.

These cases are representative of the results of this technique of combined qualities of radiation for similar lesions, and is the outline now followed routinely for these types of cases. The effect of greater doses of unfiltered radiation is too caustic and superficial in character and is not adaptable to the destruction of outlying cells without extensively destroying normal tissues with a resulting great delay in healing and a greater danger of not curing the primary lesions, because the deeper lying cells become enmeshed in the extensive fibrosis so compatible with the caustic effects of the soft or long wave length radiations and therefore tends to growth restraint or recurrence with frequent necrosis and infection.

A B

FIG. 5. Case V. Squamous cell carcinoma, Group I. Complete clinical regression for two years; treatment 10 S.E.D. (6,000 r) administered within six weeks. See details in text. A—December 12, 1928; B—June 12, 1929.

A B

FIG. 6. Case VI. Complete clinical regression eight months. Treatment: 10 S.E.D. with combined qualities of radiation. See text for details. A—February 17, 1931; B—June 27, 1931.

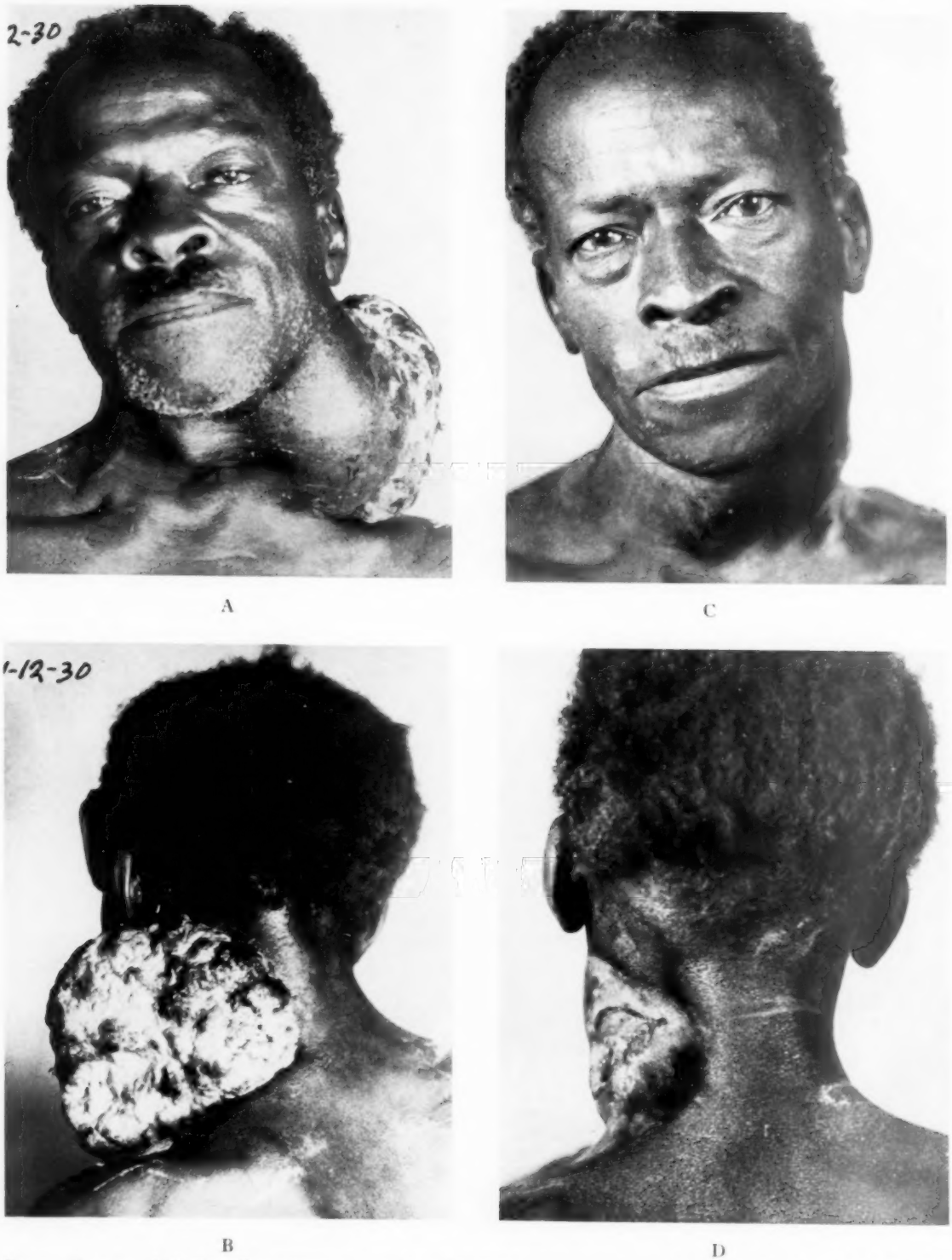


Fig. 7. Case VII. Spindle cell sarcoma; regression of about 80 per cent of neoplasm within six weeks following a total dose of 35 S.E.D. (17,500 r). See details of treatment in text. A and B—November 12, 1930; C and D—January 22, 1931.

Case VII (Fig. 7). Spindle cell sarcoma 10×14 cm. and 9 cm. thick at center and 4–5 cm. elevation of fungation about margins was treated with combined unfiltered roentgen rays and radium. It grew slowly but steadily larger after high voltage roentgen therapy. Several months later treatment was again resumed with combined unfiltered (127 kv.) and filtered high voltage (200 kv., 0.5 mm. Cu) roentgen rays and radium packs. The combined filtered roentgen rays and radium were applied to the intact skin area on the anterior and posterior surfaces of the neck and on the opposite side of the neck with a total combined dose of 2.5 S.E.D. given in divided periods over a month to each skin part. Unfiltered roentgen rays over the surface of the lesion was applied three times at weekly intervals as follows: (*a*) Four triangular areas 5×6 cm. were given 5 S.E.D. each (total 20 S.E.D., or 10,000 r). (*b*) One week later the surface of the lesion regressed to 8 cm. diameter which was divided into two areas and given 5 S.E.D. each (total 2,500 r). (*c*) At the third week the lesion regressed to 5 cm. diameter and a single 5 S.E.D. was again applied. The total dose for any given part of the surface was 15 S.E.D. (7,500 r). The total dose of unfiltered roentgen rays cross-firing through the tumor was 35 S.E.D., or 17,500 r. The additional short wave length radiation (high voltage roentgen rays and radium) admitted an additional 2.5 S.E.D. (2,000 r). The infiltration at the base of the lesion never completely subsided. The patient was remarkably comfortable for four months after complete regression of the fungating mass.

It is not fair to assume any selectivity to long wave length radiation in this instance. The failure of the previous short wave length radiation can properly be ascribed to insufficient irradiation in the light of a very resistant cellular type of cancer. The phenomenal regression should be interpreted as an effect of the caustic action of the long or soft unfiltered roentgen rays. No deleterious skin effects were noted within a period of four months.

Case VIII (Fig. 8). Adenocarcinoma of buttocks; no primary rectal disease found. Involvement diffuse, 10×15 cm., with multiple ulcerating surface nodules. There was marked swelling of entire buttocks with a deep redness of skin, suggesting a so-called malignant cellulitis.

Unfiltered roentgen radiation was applied to an area 10×15 cm. The total dose was 4 S.E.D. (2,000 r) and was repeated in three weeks. Three months later an additional 5 S.E.D. was applied. At the same time high voltage roent-

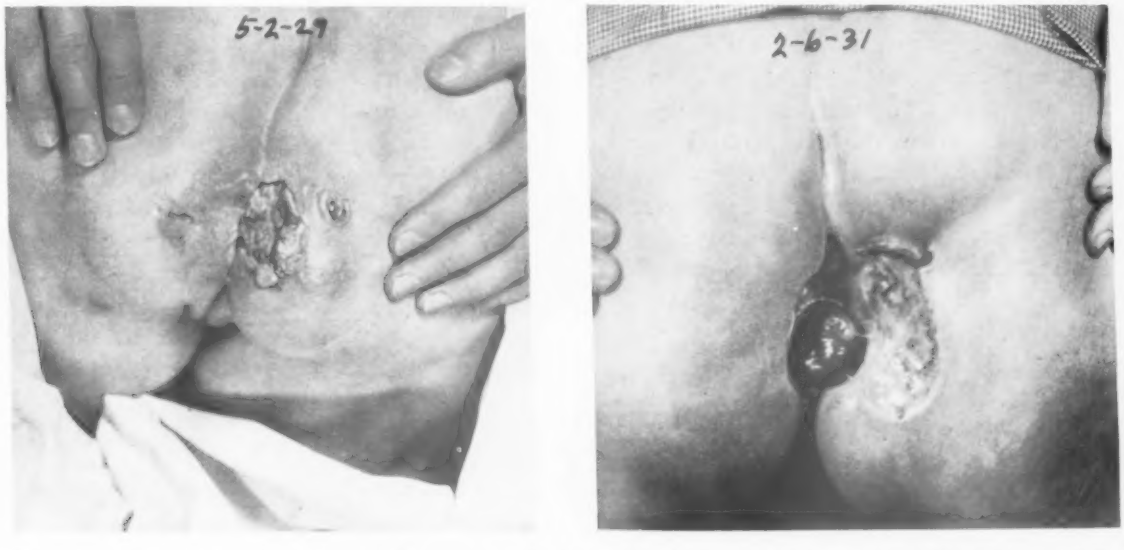


Fig. 8. Case VIII. Adenocarcinoma of buttocks. Marked clinical regression and very decided arrest of disease for a period of about three years. General health excellent at this time. Anal margin completely destroyed by disease. Treatment: 13 S.E.D. unfiltered roentgen rays administered within three months, combined with 480 per cent S.E.D. of high voltage roentgen rays during a period of five months over a field 12×16 cm.

gen radiation was applied, extending well beyond the clinically involved area as follows: May, 1929, total intensity on skin was 166 per cent S.E.D. (200 kv., 0.5 mm. Cu); July, 1929, 200 per cent with 1.5 mm. Cu; November, 1929, 120 per cent, with 0.5 mm. Cu.

Within a period of five months a total roentgen radiation was applied as follows: 13 S.E.D. (6,500 r) of unfiltered roentgen rays; 480 per cent S.E.D. (3,840 r) of high voltage roentgen rays. The area of disease rapidly regressed, leaving a deep crater-like wound with healthy base. There is no slough nor skin reaction to indicate any caustic effects of the radiation. During the past year at different times fragmentary nodular recurrences about the skin margin have been successfully controlled with gold seed implantations in isolated areas.

This case is remarkable because the patient has never ceased to continue to carry on her household duties; she has never been hospitalized and is unusually well physically over two years. The puckering of the rectal folds in the illustration presents a knob-like projection resembling a cervix uteri. The anal sphincter is destroyed and the rectum retracted.

CONCLUSIONS

There is abundant clinical evidence to indicate a wide range of technical procedures that may be successfully employed in radiation treatment of superficial skin cancers.

The difference between the minimum and maximum safe doses (250–2,500 r) are factors to be controlled by an experienced radiologist. They also depend upon the cell type of the cancer, amount of previous radiation, as well as the extent and anatomical site involved.

Dosage factors have been established at the Philadelphia General Hospital by Weatherwax as follows: 1 S.E.D. equals 500 r with unfiltered roentgen rays (127 kv.); 1 S.E.D. equals 800 r with filtered roentgen rays (200 kv., 0.5 mm. of copper). All measurements made in air.

Clinically and experimentally the biological effects of long and short wave length rays are similar. The difference is one of absorption and penetration and these factors argue for short wave length radiation in the treatment of deep-seated or infiltrating cancers. In our own clinical experience epilation and erythema factors vary with different qualities of rays. Failla found a distinct difference in effectiveness of roentgen and gamma rays for some biological effects.

All radiation procedures should be carefully checked with ionization and half-value layer estimations.

Small superficial cancers (1 to 2 cm. in diameter), in our experience, are best treated by electrodesiccation, after biopsy, to be followed by curettement of the charred base so that no soft bleeding granulations of malignancy will be left intact. A localized dose of 2 S.E.D. (127 kv.), no filter (1,000 r) or 2 mm. of aluminum (1,100 r) usually completes an adequate treatment for a basal cell cancer.

If biopsy shows squamous cell cancer then additional radiation 2 S.E.D. (1,100 r), (127 kv. 2 mm. Al) localized to the lesion should be given one week later. Gamma radium (2 to 4 mm. brass filter) is also widely and successfully used (with electrodesiccation 2 S.E.D. and without electrodesiccation about 2 to 4 S.E.D.). High voltage roentgen rays should routinely be given to the glandular area of the neck to the limit of skin tolerance (150 per cent S.E.D. in about two weeks).

Unfiltered roentgen rays for the small lesions should not exceed 2 S.E.D. This dose may be repeated once a week for two to three weeks. Heavier doses at more frequent intervals have been given with success but such intensities cannot be prescribed nor recommended because the latitude of danger is so small and the procedure is almost entirely empirical and may fail even with the most expert operator. The procedure has an additional handicap from the anxiety caused by the sluggish regressions that are frequently encountered (six to eight weeks). Telangiectatic scars with thickening are sometimes unsightly and are always potential areas for secondary malignant degeneration.

The use of filtered roentgen rays or radium alone needs no discussion. Their curative value with good results is well known and except for lesions about the eyelids, must, in our experience, rank second to electrodesiccation, to be followed by roentgen rays or radium.

The use of unfiltered roentgen radiation has been followed by excellent regressions and cures for large (2.5 to 15 cm. diameter) superficial cancers, but the good results appear to be entirely dependent upon the caustic effects of the long or soft radiation. There is no safe mathematical procedure for estimating the necessary intensity of unfiltered roentgen rays. As much as 10 to 15 S.E.D. has been administered with excellent clinical regressions when the tumor thickness was more than 1 cm. A thickness of 5 to 10 mm. can be given 5 to 10 S.E.D., but it is safer to give this in divided doses over a period of two to three weeks.

Squamous cell cancers should always be supplemented with gamma radium rays to the surrounding healthy margins as well as the actual lesion because the cellular arrangement is less compact than the basal cell cancer, and tends to ramify into the surrounding normal structure to an extent that is not amenable to clinical recognition.

For this reason high voltage roentgen radiation or radium in the form of packs must also be given to the surrounding glandular regions. Ordinarily about 6 to 8 S.E.D. (3,000–4,000 r) of unfiltered roentgen rays in divided doses (2 S.E.D. once a week) localized to the lesion, combined with 2–4 S.E.D. (1,500–3,200 r) of gamma radium rays on the lesion are effectual for infiltrating inoperable cancers of wide involvement, particularly about the lip.

This experience clearly demonstrates that there is no mathematical procedure that can be set down for the administration of great intensities of unfiltered roentgen rays. A full account of the technique and experience with large doses is given for the various cases, illustrating a wide latitude of doses employed. Radiation necrosis has not been a serious factor in our experience but this element is great if special precautions are not exercised. Only large superficial cancers—2.5 to 10 cm. in diameter—should receive these intensities of unfiltered roentgen rays.

The results are sufficiently striking to justify the recommendation of unfiltered roentgen rays for selected cases of wide involvement, and to be combined with heavily filtered radium and roentgen rays for squamous cell cancers.

REFERENCES

1. CHAMBERLAIN, W. E., and NEWELL, R. R. The epilation and erythema dose; comparison of filtered and unfiltered roentgen ray. *Radiology*, 1928, *10*, 280–283.
2. ERSKINE, A. W. Practical X-Ray Treatment. Bruce Publishing Company, St. Paul, 1931.
3. FAILLA, G., and HENSHAW, P. S. The relative biological effectiveness of x-rays and gamma rays. *Radiology*, 1931, *17*, 1–43.
4. GLASSER, O., and MEYER, W. H. Erythema dose in roentgen units. *Strahlentherapie*, 1926, *23*, 361.
5. MARIE, T. The value of measurements of the intensity of radiation in establishing rational radiation therapy. *J. Cancer Research*, 1927, *11*, 377–416.
6. MARTIN, J. M. Radiation therapy in treatment of cancer of the mouth and lips. *Radiology*, 1931, *16*, 881–892.
7. MEYER, W. H., and GLASSER O. Erythema doses in absolute units. *Radiology*, 1926, *6*, 320–325.
8. POHLE, E. A., and BARNES, J. M. Clinical and physical investigations on the problem of dosimetry in roentgen therapy. *Radiology*, 1928, *10*, 300–311.
9. ULLMANN, H. J. Discussion of paper by Martin, ref. 6.
10. WEATHERWAX, J. A., and Robb, C. To be published.
11. WIDMANN, B. P. Radiation technic for cancer of the mouth with combinations of gamma radium rays and varying qualities of high voltage roentgen rays. *Radiology*, 1930, *14*, 197–206.
12. WOOD, F. C. Effect on tumors of radiation of different wave lengths. AM. J. ROENTGENOL. & RAD. THERAPY, 1924, *12*, 474–482.

DISCUSSION

Dr. A. U. Desjardins, Rochester, Minn. I must say I admire Dr. Widmann's courage, because it takes a good deal of intestinal fortitude to tackle such a scheme of treatment. Probably the main secret of his fortitude is the character of the patients he is dealing with, mostly old persons with hopeless lesions. Any effort to make their life more comfortable is worth while. With the class of patients with whom we have to deal we could not, as a rule, go quite so far. But in selected cases, I have followed a somewhat similar scheme. I learned years ago that a heavy, single dose of unfiltered or filtered rays over a small area may be given without fear of ulceration, but if the area is too large, ulceration is likely. This is not an infallible, but a general rule.

On the other hand, in dealing with lesions larger than 5 centimeters, filtered rays are preferable. To a small area, from three to five erythema doses may be given, and this may be followed at the same session by from three to five erythema doses of unfiltered rays. It is amazing to see the regression that takes place, sometimes within a week.

In many such cases the patients are made much more comfortable, the discharges are diminished greatly or sometimes disappear completely for a time, and the problem of dressings is greatly simplified. They ultimately die from metastasis or from some complication. Dr. Widmann has already emphasized that it is almost impossible to standardize such a scheme of treatment so that all radiologists may follow a particular formula. Each case is a distinct problem. I make it a point to select such cases myself and help the technician plan the fields and arrange the lead protection so that it fits the lesion exactly. I set the tube for the technician; all she has to do is to push the button and watch.

Vol. XXVIII, No. 4

A NOTE ON SHADOWS OF FENESTRATED RIBS IN ROENTGENOGRAMS*

By HENRY C. SWEANY, M.D.
CHICAGO, ILLINOIS

THE anomalous development of anatomic structures is only one of the extreme variations that organic life is heir to. Some of these anomalies are important to surgeons, some to biologists, while certain

FIG. 1. Roentgenogram of patient W. O., clearly showing a fenestrated right third rib beneath an upper lobe involvement.

ones are of interest to roentgenologists. Although anomalous ribs are relatively common, they sometimes cause difficulty in the interpretation of roentgenograms, especially where they form annular shadows that may simulate cavities, pleural blebs, etc. The incidence of rib anomalies is from 1 to 2 per cent, but the fenestrated or forked ribs causing annular shadows are fewer in number, although perhaps not as few as the scarcity of reports might lead us believe.

In 1884, Ferrier[1] described a bifurcated third left costal cartilage forming an opening 3.5 by 2.5 cm. Obviously there was no mention made of roentgen shadows at that time, but the author considered such anomalies rare because he had not found any reports in the literature. No doubt they were more difficult to find than after the roentgen ray was made use of. In 1899, Popowsky[2] presented a series of 4 such bifurcated ribs, viz., the second, third, and fourth ribs on the right, and the fourth rib on the left. All were connected with the sternum by cartilage junctions. The author considered it a regressive phenomenon because in anthropoids and human embryos there are eight ribs connected to the sternum which is arranged in eight pairs of

FIG. 2. Same as Figure 1, ten months later, with a more extensive involvement of an abscess on a moderately advanced tuberculosis, giving an impression of a definite annular shadow.

segments. Ruppricht,[3] in 1929, reported a similar bifurcation of the right third and fourth in a fetus. He mentions another adult specimen in the Anatomical Institute of Vienna where there were three bifurcated ribs on the right side, and mentions

* From the Research Laboratories of the Municipal Tuberculosis Sanitarium, Chicago, Illinois.

Fig. 3. A sagittal section of the lung showing small fibrotic cavities in the upper lobe, but little lung involvement beneath the annular shadow.

the fact that bifurcation is not uncommon.

Where there are pathological changes in the viscera, such as the consolidation or excavations of tuberculosis, these anomalies become a problem for interpretation. In our experience we have found that the fenestrated third and fourth ribs produce annular shadows that may interfere with the interpretation of hilar shadows, especially on single plates. Of 9 such anomalous ribs found in nearly 2,500 patients, 6 were on the third and fourth ribs where they interfere greatly with tuberculous lung shadows. There were 2 in females and 7 in males; 3 were on the third right; 3 on the fourth left; 2 on the fifth right; one on the fifth left and one on the seventh left. Although I do not claim to have found all such anomalies where the field is hidden by dense fibrosis or infiltration, raised diaphragm, etc., enough has been observed to show that the greater number are found on the third right and the fourth left where they would tend to interfere most with hilar shadows.

Fig. 4. Another patient, W. S., with a similar fenestrated third rib, shown after removal at autopsy in Figure 5.

Fig. 5. Right third rib, removed from patient W. S., shown in Figure 4.

We were fortunate in being able to follow one patient from the early stages of a superimposed abscess on a tuberculous lung, to the autopsy, and have illustrated it in Figures 1, 2 and 3. In Figure 1 the fenestrated rib shows plainly because the pathology is slight. In Figure 2, however, the annular shadow is confusing, but by comparison with a sagittal lung section there is no lung cavity beneath. There are, however, several small cavities nearer the periphery and towards the apex.

The upper fork of the rib seems to be immediately over the interlobar fissure and intensifies its density. The result is a definite annular shadow surrounding a field of

Fig. 6. A roentgenogram of a patient, C. G., having a fenestrated right third and a forked left fourth rib.

relatively normal lung markings. Inasmuch as thin-walled cavities or pleural blebs and bullae may present the same appearance, the interpretation of such shadows may not always be easy. Figure 4 is a roentgenogram showing a fenestrated right third rib, and Figure 5 shows this rib after removal at autopsy. The confusion in the roentgenogram is evident. Figure 6 shows a broad fenestration of the third right with an anomalous second left, also a forked left fourth rib in the same specimen. Figure 7 is a fenestrated fourth left removed at autopsy from a female patient. This was entirely overlooked before the autopsy, due chiefly to advanced tuberculosis pathology beneath it.

Fig. 7. A photograph of a fenestrated fourth rib from a woman.

SUMMARY

A report is made of observations on fenestrated and forked ribs in tuberculous patients with emphasis on the interpretation of such shadows in roentgenograms. Although perhaps low, due to the difficulty of making observation on tuberculous patients, an incidence of 0.4 per cent was found in nearly 2,500 patients. Seven out of 9 were in men, and one was bilateral. The third right and the fourth left are the most frequently involved.

REFERENCES

1. Ferrier. Bifurcation of a costal cartilage; supernumerary intercostal muscle. *Bull. Soc. d'anat. et physiol. de Bordeaux*, 1884, *5*, 34–35.
2. Popowsky, J. Contribution to the question of division of ribs. *Anat. Anz.*, 1899, *15*, 284–288.
3. Ruppricht. Two bifurcated ribs in a fetus. *Ztschr. f. d. ges. Anat.*, 1929, *91*, 402.

A THYRATRON PEAK VOLTMETER*

By CHARLES WEYL, S. REID WARREN, JR., *and* C. JUSTUS GARRAHAN
PHILADELPHIA, PENNSYLVANIA

Abstract. An accurate, simple and convenient peak voltmeter, employing a thyratron, for the measurement of roentgen-ray tube peak voltages is described. This instrument, in conjunction with a 100 megohm resistor, is intended to be used as an improved substitute for the sphere gap.

INTRODUCTION

IN a recent paper[1] a vacuum tube peak voltmeter which it is believed has a number of advantages over the sphere gap was described in detail. Since that paper was written an improved peak voltmeter, using a thyratron has been designed, tested and in constant use for more than six months in our laboratory.

THE THYRATRON

The thyratron is a three electrode device. The electrodes are arranged in a manner similar to those of the three electrode vacuum tube, with hot cathode and anoed separated by a metal mesh called the grid. After evacuation of the thyratron bulb, a small amount of mercury is introduced into the bulb before sealing off.

A comprehensive analysis of thyratron operating characteristics is very complicated.[2] A few facts which may be determined experimentally will suffice for the explanation of the peak voltmeter to be described in the next section. The tube may be connected as shown in Figure 1.

If the input leads are short circuited, and *S* is closed, it will be found that as the negative grid bias is decreased gradually (grid brought nearer to cathode potential), the anode-cathode current will start suddenly at a well-defined grid-cathode voltage. At this point, the anode-cathode current rises abruptly from zero to a value determined by the anode battery voltage, the load resistance and the anode-cathode resistance.

[1] Weyl, C., Warren, S. R. Jr., and McPhedran, F. M. Methods of measuring and recording roentgen-ray tube currents and voltages. Am. J. Roentgenol. & Rad. Therapy, 1931, *26*, 871–877.

[2] Hull, A. W. Hot cathode thyratrons. *General Electric Rev.*, April, 1929, p. 213; July, 1929, p. 390.

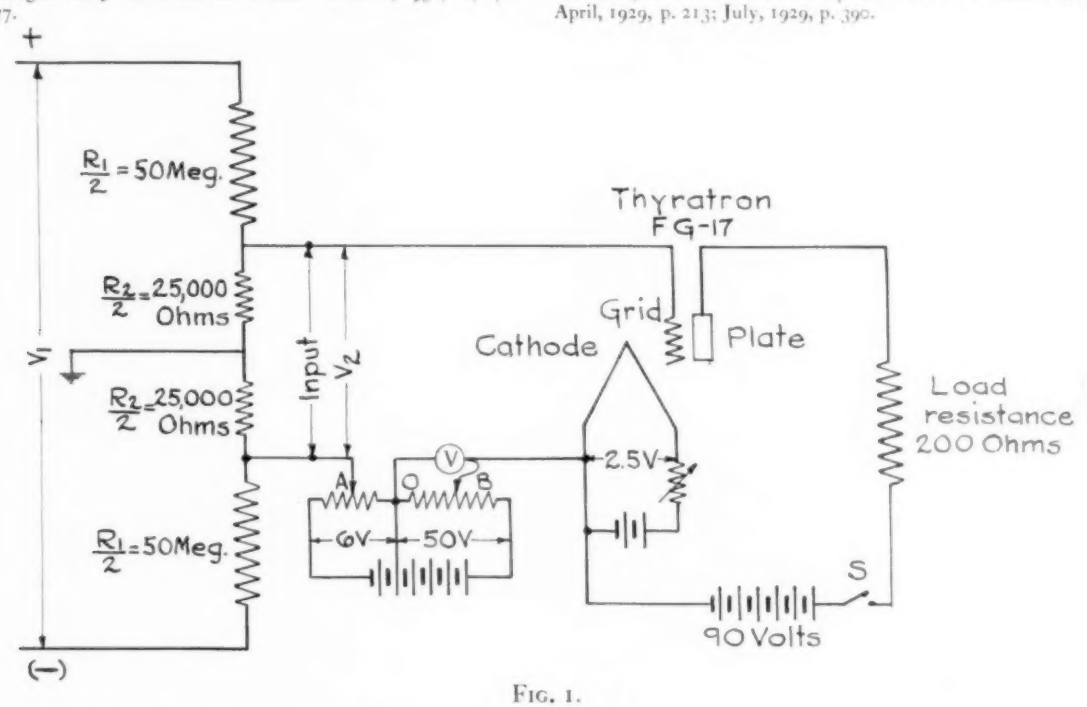


FIG. 1.

* The work described has been conducted at the Moore School of Electrical Engineering, University of Pennsylvania, under a grant by the National Tuberculosis Association.

The value of this anode-cathode current is *not* dependent on subsequent positive or negative values of grid-cathode voltage. The anode current flow is associated with a blue discharge of ionized mercury vapor throughout the bulb of the thyratron. When the arc is started, switch S must be opened to break it. *The grid potential controls only the starting of the arc.*

The anode-cathode resistance of the arc is less than 50 ohms during normal operation. Care must be taken that a suitable load resistance be connected in the anode circuit to limit the current to a value not destructive to the hot cathode.

When it is desired to use the thyratron for accurate measurements it is necessary to allow the cathode to preheat about five minutes to attain equilibrium of temperature.

The particular thyratron employed for the instrument described below is designated Type *FG*-17. The following data are quoted from the manufacturers' instructions:[3] cathode: volts—2.5, amperes—5.0; anode current: amperes—0.5; anode-cathode voltage drop: maximum—24 volts, minimum—10 volts; approximate starting characteristics: anode voltage +100 volts; grid voltage −2.25 volts. Mounting—standard four prong base; overall length—6 1/2 inches; diameter—2 7/16 inches.

THE PEAK VOLTMETER

If a fixed resistance of 100 megohms, non-inductive and with low distributed capacitance be connected across the anode and cathode of a roentgen-ray tube employed with single phase roentgenographic apparatus of which the midpoint is grounded, the instantaneous value of the current through the resistance will be proportional to the voltage applied to the roentgen-ray tube at the same instant.[2] A device for measuring the peak value of the voltage V_2 across a *part* of the 100 megohm resistance makes possible the calculation of the peak voltage V_1 applied to the roentgen-ray tube. For a short roentgenographic exposure the voltage V_2 will be of some form such as that shown in Figure 2. The reading of the thyratron peak voltmeter indicates the peak value of V_2 for this exposure. Then the peak value of the roentgen-ray tube voltage $\frac{V_1 = R_1 + R_2}{R_2} V_2$.

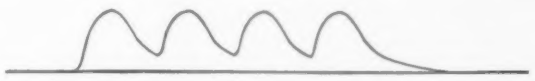


FIG. 2. Oscillogram of V_2 taken by means of a linear vacuum tube amplifier and oscillograph Roentgen-ray tube peak voltage—65 kv. peak.

The procedure for measuring the peak value of V_2 with the thyratron peak voltmeter connected as shown in Figure 1 is as follows: With the slide of potentiometer B set at position O, the slide of potentiometer A is moved to the point nearest O at which no discharge occurs in the thyratron. This discharge consists of a visible blue glow throughout the bulb occurring while anode-cathode current flows. This discharge provides a definite signal that the grid potential has a value at or below the starting point of the thyratron characteristic. Therefore the adjustment described above has brought the grid to a potential such that any small positive increment of voltage applied to the input will cause the discharge to occur.

The actual procedure of adjustment is as follows: With the switch S closed the slide of the potentiometer A is moved slowly towards O from a point at which no discharge occurs to a point at which the discharge just begins. The switch S is then opened. The slide is moved the smallest possible distance away from O. The switch S is reclosed. If the glow appears switch S is again reopened and the slide is again removed minutely from O. This operation is repeated until the slide is at the point *nearest* O at which the glow does not appear when the switch S is closed. The above procedure adjusts the voltmeter to zero.

With the switch S closed, the slide of the potentiometer B is brought to a point such that the O-50 voltmeter V reads near full scale and a short exposure is made through the roentgen-ray tube.

[3] Instructions GEJ-278C, General Electric Company.

Potentiometer B is then adjusted carefully by successive approximations in the same manner as described for the adjustment of potentiometer A to the point where the thyratron just starts discharge when the exposure is made.

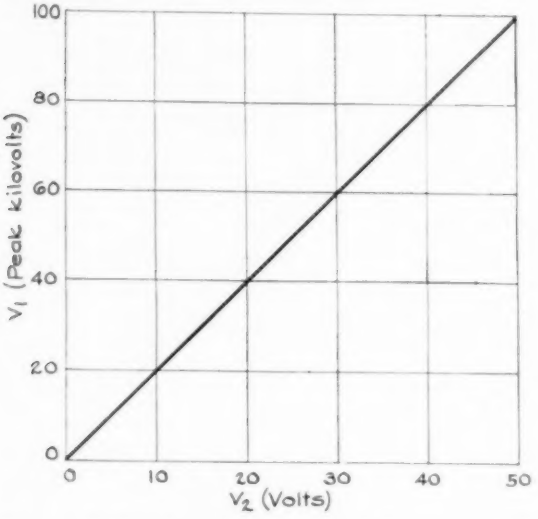


FIG. 3. Calibration of resistor and thyratron peak voltmeter for $R_1 = 100$ megohms; $R_2 = 50{,}000$ ohms. Roentgen-ray tube voltage is V_1; input voltage to thyratron voltmeter is V_2.

The reading of the voltmeter V for this final adjustment of the potentiometer B is the direct measure of the peak value of V_2 for this exposure. Values of V_2 may be plotted against V_1 (Fig. 3) to give a quick reference between voltmeter readings and roentgen-ray tube peak kilovoltage values.

This equipment may be used at ground potential provided the roentgenographic apparatus is so connected that the center point of R_2 is always at ground potential during the exposure. If an alternating current milliammeter (O–1 ma.) connected from the mid-point of R_2 to ground reads zero during exposure, the peak voltmeter and resistor may be used as described, the voltmeter at ground potential.

With a little practice the adjustments of the potentiometer may be made with rapidity and accuracy. The simplicity and reliability of operation and the accuracy of measurement with this instrument have been found superior to any high tension peak voltmeter arrangement tried in our laboratory.

APPENDIX

Oscillographic Record of Action of Thyratron Peak Voltmeter

A method for recording the voltage V_2 by means of a vacuum tube amplifier and oscillograph has been described.[1]

To test the action of the peak voltmeter one element of a two element oscillograph is connected to record V_2 and a second element is connected, in parallel with a shunt, in series with the load resistance of the thyratron peak voltmeter. The simultaneous record of V_2 and the thyratron anodecathode current indicates the accuracy with which the voltmeter may be adjusted.

Figure 4 is a tracing of one of these oscillograms. The circuit employed was arranged in such a way that after the starting of the thyratron the record of V_2 is obliterated by the superposition of a direct voltage resulting from direct current flowing in the grid circuit of the thyratron. Therefore Figure 4 shows that for 1/240 second the voltage

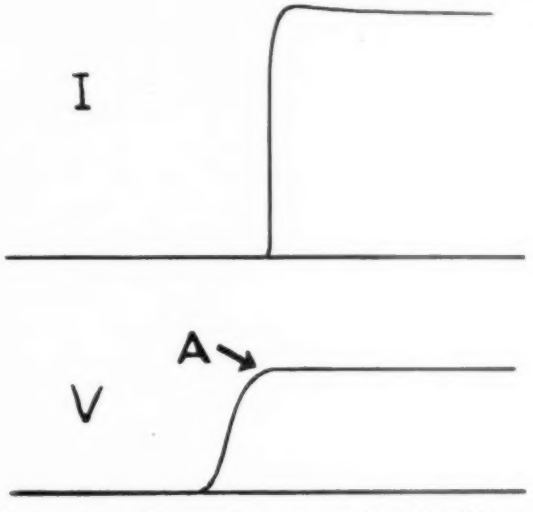


FIG. 4. V-Oscillographic trace of V_2. I-Oscillographic trace of thyratron anode-cathode current. Connections as in Figure 1. V and I photographed simultaneously.

V_1 rises from zero to 65 kv., the peak value. At the point A—65 kv. the thyratron operates. The setting for potentiometer B is such that voltmeter V reads 32.5 volts. The value $V_1 = 65$ kv. is obtained from the calibration curve (Fig. 3).

Vol. XXVIII, No. 4

THE AMERICAN JOURNAL OF ROENTGENOLOGY AND RADIUM THERAPY

Editor: Lawrence Reynolds, M.D.

Editorial Board: A. C. Christie, M.D. E. H. Skinner, M.D. Lauriston S. Taylor

Advisory Board for Pathology: James Ewing, M.D. Eugene L. Opie, M.D.

Collaborating Editors: The Officers and Committee Members of the Societies of which this Journal is the official organ, whose names appear on this page, are considered collaborating editors of this Journal.
Foreign Collaborators: A. Béclère, M.D., Paris, Gösta Forssell, M.D., Stockholm, G. F. Haenisch, M.D., Hamburg, R. Ledoux-Lebard, M.D., Paris.

Publisher: Charles C Thomas, 220 East Monroe St., Springfield, Illinois

Issued Monthly. Subscription $10.00 per year, $11.00 in Canada and $12.00 in foreign countries. Advertising rates submitted on application: Editorial office, 110 Professional Building, Detroit, Mich. Office of publication, 220 E. Monroe St., Springfield, Ill. Information of interest to all readers will be found on page iv.

Officers and Standing Committees

THE AMERICAN ROENTGEN RAY SOCIETY

President: William A. Evans, Detroit, Mich.; *President-Elect:* John T. Murphy, Toledo, Ohio; *1st Vice-President:* Sherwood Moore, St. Louis, Mo.; *2d Vice-President:* Robert Drane, Savannah, Ga.; *Secretary:* E. P. Pendergrass, University Hospital, Philadelphia, Pa.; *Treasurer:* E. L. Jenkinson, 1439 S. Michigan Ave., Chicago, Ill.; *Historian:* E. H. Skinner, Kansas City, Mo.

Executive Council: C. A. Waters, Chairman, 1100 N. Charles St., Baltimore, Md., M. C. Sosman, Boston, Mass., C. L. Martin, Dallas, Texas, W. A. Evans, Detroit, Mich., J. T. Murphy, Toledo, Ohio, L. Reynolds, Detroit, Mich., E. P. Pendergrass, Philadelphia, Pa., E. L. Jenkinson, Chicago, Ill.

Committee on Laws and Public Policy: B. R. Kirklin, Chairman, Rochester, Minn., F. M. Hodges, Richmond, Va., W. E. Chamberlain, Philadelphia, Pa.

Committee on Safety and Standards: H. K. Pancoast, Philadelphia, Pa., W. D. Coolidge, Schenectady, N. Y., A. U. Desjardins, Rochester, Minn., H. J. Ullmann, Santa Barbara, Calif., B. H. Nichols, Cleveland, Ohio, G. E. Richards, Toronto, Canada, R. R. Newell, San Francisco, Calif.

Publication Committee: Henry K. Pancoast, Chairman, Philadelphia, Pa., B. R. Kirklin, Rochester, Minn., Ross Golden, New York City.

Tube Committee: David R. Bowen, Chairman, Philadelphia, Pa., I. H. Lockwood, Kansas City, Mo., E. C. Ernst, St. Louis, Mo., G. W. Grier, Pittsburgh, Pa., E. A. Pohle, Madison, Wis.

Finance Committee: G. W. Grier, Chairman, Pittsburgh, Pa., J. Bennett Edwards, Leonia, N. J., Leslie R. Hess, Hamilton, Ont.

Committee for American Congress of Radiology, Chicago, 1933: E. L. Jenkinson, Chairman, Chicago Ill., Lawrence Reynolds, Detroit, Mich., J. T. Murphy, Toledo, Ohio.

Member, National Research Council: W. F. Manges, Philadelphia, Pa.

Editor: Lawrence Reynolds, 110 Professional Building, Detroit, Mich.

Editorial Board: A. C. Christie, E. H. Skinner, Lauriston S. Taylor.

Advisory Board for Pathology: James Ewing, Eugene L. Opie.

Thirty-fourth Annual Meeting: Chicago, Ill., Sept. 25–30, 1933.

THE AMERICAN RADIUM SOCIETY

President: Burton J. Lee, New York City; *President-Elect:* Rollin H. Stevens, Detroit, Mich.; *First Vice-President:* G. W. Grier, Pittsburgh, Pa.; *Second Vice-President:* Howard Morrow, San Francisco, Calif.; *Secretary:* William H. Cameron, 511 Fifth Ave., New York City; *Treasurer:* Zoe A. Johnston, Jenkins Arcade, Pittsburgh, Pa.

Executive Committee: H. H. Bowing, Chairman, Rochester, Minn., H. J. Ullmann, Santa Barbara, Calif., Sanford Withers, Denver, Colo.

Program Committee: B. J. Lee, Chairman, Memorial Hospital, New York City, E. H. Skinner, Kansas City, Mo., F. E. Simpson, Chicago, Ill.

Publication Committee: E. H. Skinner, Chairman, Kansas City, Mo., B. F. Schreiner, Buffalo, N. Y., Douglas Quick, New York City.

Research and Standardization Committee: G. Failla, Chairman, New York City, H. J. Ullmann, Santa Barbara, Calif., R. B. Greenough, Boston, Mass.

Education and Publicity Committee: G. E. Pfahler, Chairman, Philadelphia, Pa., C. L. Martin, Dallas, Texas, J. S. Ullman, Natchez, Miss.

Committee for American Congress of Radiology, Chicago, 1933: James T. Case, Chairman, Chicago, Ill., Albert Soiland, Los Angeles, Calif., Henry Schmitz, Chicago, Ill.

Eighteenth Annual Meeting: Chicago, Ill., Sept., 25–30, 1933.

EDITORIAL

THE ANNUAL MEETING

IT HAS been thirty-three years since the organization meeting of the American Roentgen Ray Society in St. Louis in 1900 and during this period enormous progress has been made in every field of roentgenology as evidenced by the splendid program which was arranged by Dr. William A. Evans, President-elect, for the Thirty-third Annual Meeting of the Society held in Detroit, Michigan, September 27 to 30, 1932. Dr. Evans' plan for the program was that special attention should be given to the clinical aspect of roentgenology. That he succeeded in this is indicated by the diversity of the program and in the various symposia which were arranged. These included a symposium on "Joint Pathology"; a symposium on "Industrial and Medico-legal Practice"; a comprehensive symposium on "Tuberculosis" including its clinical, its diagnostic and therapeutic phases and the importance of the roentgen ray in the various aspects of the tuberculosis problem; a symposium on "Gynecology and Obstetrics" which included such comprehensive topics as the "Pituitary Function as Related to Certain Gynecological Problems" and "Roentgen Study as an Aid in Obstetrics." There were also a number of papers dealing with the radiation therapy of malignant diseases. The Society was extremely fortunate in having among its guests Dr. C. C. Little, President of the American Society for the Control of Cancer, who discussed "The Relation of the American Society for the Control of Cancer to Roentgenologists" and who also took an important part in the discussion of radiation therapy from its biological aspect. Of unusual interest were the group of papers given by the Staff of the Memorial Hospital on "The Relative Effects Produced by 200 kv. X-rays, 700 kv. X-rays and Gamma Rays." One of the outstanding symposia of the entire meeting was that on "Malacic Diseases of Bone" conducted by Dr. Max Ballin and Dr. Plinn F. Morse, whose outstanding contributions on malacic diseases are well known. This subject was discussed under four main headings: (a) The Clinical Symptomatology of Osteomalacias (including Calcium and Phosphorus Metabolism, Muscular Hypotonia); (b) Pathologic Classification; (c) Etiologic Classification (Hereditary, Nutritional, Endemic and Endocrine Factors); (d) Roentgenologic Grouping. The last-named group included rickets, osteomalacia, primary hyperparathyroidism, osteitis fibrosa cystica, leontiasis ossea, giant cell sarcoma, ankylosing spondylitis and polyarthritis, osteopoikilosis (doubtful), marble bone disease, Kashin-Beck's disease, renal rickets, osteogenesis imperfecta, decalcification (in other endocrine disturbances), Christian's syndrome, Cooley's syndrome, and also a paper on "Irradiation of the Parathyroids in Cystic Disease of the Bones."

The Caldwell Lecture, the thirteenth since its foundation, was delivered by Dr. A. W. Crane of Kalamazoo, Michigan, who chose as his subject "Roentgen Findings and the Clinical Background." Dr. Crane could not have chosen a more timely subject for the entire program from beginning to end illustrated to a marked degree the importance of a clinical background as the basis for roentgenology. Never in the whole history of roentgenology has the importance of a clinical background been more imperative than at the present when roentgenology touches every phase of medicine, and the roentgenologist of the present and future must not only be a master tech-

nician in every sense that this term implies but he must also fortify himself with a knowledge of the clinical aspects of medicine if he is to succeed and if roentgenology it to maintain its present high standard.

The lecture on Tuesday evening by Professor George L. Clark of the University of Illinois, on "The X-ray in Industry and other Non-medical Fields" was both interesting and instructive. He first discussed industrial roentgenography illustrating the various uses by numerous excellent slides with special attention to the detection of flaws in heavy castings and checking of welding of high pressure tanks. He followed with a description of the method of study of submicroscopic structure by the diffraction method by which every metal and alloy is shown to have its characteristic crystal structure. He demonstrated that deformation by industrial processing could be accurately followed by this method, thereby enabling the determination of the physical characteristics of the product. Lastly, he indicated that material supposedly non-crystalline in structure, such as pearls and other animal tissues, can be studied by the diffraction method and certain changes therein induced by physical and chemical influences resulting in variations in structure detected and determined in terms of molecular structure. It was predicted that research in tissue study may in time reveal valuable information of diagnostic and clinical importance.

Mention should also be made here of the completeness and the value of the Scientific and Commercial Exhibits. They furnished a source of unending interest during the meeting. A more detailed account of these exhibits will be found elsewhere in this issue of the Journal.

A contributing feature to the success of the meeting can be ascribed to the disposition of the various rooms given over to the meeting at the Book-Cadillac Hotel. The Society wishes through its Journal to thank the management of the Hotel for their courtesy and many kindnesses extended to members and guests throughout the meeting.

The following officers were elected for the coming year: *President:* Dr. William A. Evans; *President-Elect:* Dr. John T. Murphy; *1st Vice-President:* Dr. Sherwood Moore; *2d Vice-President:* Dr. Robert Drane; *Secretary:* Dr. E. P. Pendergrass; *Treasurer:* Dr. E. L. Jenkinson; *Member of the Executive Council:* Dr. Charles L. Martin.

SOCIETY PROCEEDINGS, CORRESPONDENCE AND NEWS ITEMS

Items for this section solicited promptly after the events to which they refer.

MEETINGS OF ROENTGEN SOCIETIES*

United States of America

American Roentgen Ray Society
Secretary, Dr. E. P. Pendergrass, University Hospital, Philadelphia, Pa.
Annual Meeting: Chicago, Ill., Sept. 25–30, 1933.

American College of Radiology
Secretary, Dr. Albert Soiland, 1407 S. Hope St., Los Angeles, Calif.
Annual Meeting: Chicago, Ill., Sept. 25–30, 1933.

Section on Radiology, American Medical Association
Secretary, Dr. J. T. Murphy, 421 Michigan St., Toledo, Ohio.
Annual meeting: Milwaukee, Wis., 1933.

Radiological Society of North America
Secretary, Dr. D. S. Childs, 607 Medical Arts Bldg., Syracuse, N. Y.
Annual Meeting: Atlantic City, N. J., Nov. 28–Dec. 2, 1932.

Radiological Section, Los Angeles County Medical Society
Secretary, Dr. D. R. MacColl, 2826 S. Hope St., Los Angeles, Calif.
Meets on the third Wednesday of each month at the California Hospital.

Radiological Section, Southern Medical Association
Secretary, Dr. C. H. Heacock, 20 S. Dunlap St., Memphis, Tenn.
Annual meeting: Birmingham, Ala., Nov. 16–18, 1932.

Brooklyn Roentgen Ray Society
Secretary, Dr. J. J. Masterson, 401-76th St., Brooklyn, N. Y.
Meets monthly on first Tuesday, October to April.

Buffalo Radiological Society
Secretary-Treasurer, Dr. Joseph S. Gian-Franceschi, 610 Niagara St., Buffalo, N. Y.
Meets second Monday of each month except during summer months, place of meeting selected by the host.

Chicago Roentgen Society
Secretary, Dr. George M. Landau, 660 Groveland Park.
Meeting second Thursday of each month October to May inclusive at Virginia Hotel.

Cincinnati Radiological Society
Secretary, Dr. H. G. Reineke, Christian R. Holmes Hospital, Cincinnati, Ohio.
Meetings held monthly.

Cleveland Radiological Society
Secretary, Dr. M. A. Thomas, Medical Arts Bldg.
Meetings are held at 6:15 P.M. at the Cleveland Chamber of Commerce Club rooms on the fourth Monday of each month from October to April, inclusive.

Detroit Roentgen Ray and Radium Society
Secretary, Dr. E. R. Witwer, Harper Hospital.
Meets monthly on first Thursday from October to May, at Wayne Country Medical Society Building.

Florida Radiological Society
Secretary, Dr. W. McL. Shaw, 418 St. James Bldg., Jacksonville, Fla.
Meetings held twice a year, May and November.
Next meeting, Jacksonville, November, 1932.

Illinois Radiological Society
Secretary, Dr. L. M. Hilt, Myers Bldg., Springfield, Ill.
Regular meetings held quarterly.

Indiana Roentgen Society
Secretary, Dr. J. N. Collins, Indianapolis, Ind.
Annual meeting each February 22 in Indianapolis.

Milwaukee Roentgen Ray Society
Secretary, Dr. J. E. Habbe, 221 Wisconsin Ave., Milwaukee, Wis.
Meets first Friday in October, December, February and April.
Place of meeting designated by the president.

Minnesota Radiological Society
Secretary, Dr. L. G. Rigler, University Hospital, Minneapolis, Minn.
Next meeting, Duluth, Minn., Oct. 8, 1932.

New England Roentgen Ray Society
Secretary, Dr. Thomas R. Healy, 370 Marlboro St., Boston, Mass.
Meets monthly on third Friday, Boston Medical Library.

New York Roentgen Society
Secretary, Dr. C. W. Schwartz, 33 E. 68th St., New York City.
Meets monthly on third Monday, New York Academy of Medicine, at 8:30 P.M.

Central New York Roentgen Ray Society
Secretary, Dr. M. T. Powers, 250 Genesee St., Utica, N. Y.
Three meetings a year—January, May and November.

Pennsylvania Radiological Society
Secretary, Dr. W. E. Reiley, Clearfield, Penna.

Philadelphia Roentgen Ray Society
Secretary, Dr. Karl Kornblum, 3400 Spruce St.
Meeting first Thursday of each month from October to May inclusive, at 8:15 P.M., in Thomson Hall, College of Physicians, 19 S. 22d St.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary, Dr. Stafford L. Warren, Strong Memorial Hospital.
Meets monthly on the first Friday evening at 7:45 at the Rochester Medical Association Building.

St. Louis Roentgen Club
Secretary, Dr. W. K. Mueller, University Club Bldg.
Meets first week of each month. Time and place of meetings designated by president.

South Carolina X-Ray Society
Secretary: Dr. R. B. Taft, 105 Rutledge Ave., Charleston, S. C.
Meets at time and place of South Carolina State Medical Association. Next meeting, Spartansburg, April, 1933.

Texas Radiological Society
Secretary-Treasurer, Dr. C. P. Harris, Houston, Texas.
Meets annually one day preceding the meeting of the Texas State Medical Association.

University of Michigan Roentgen Ray Society
Secretary, Dr. C. C. Taylor, University Hospital, Ann Arbor, Mich.
Meets first and third Wednesday evening of the month from October to June, at 8 o'clock in the amphitheatre of the University Hospital.

* Secretaries of Societies not here listed are requested to send the necessary information to the Editor.

Virginia Roentgen Ray Club
Secretary, Dr. Wright Clarkson, 205 S. Sycamore St., Petersburg, Va.
Meets annually in October.
Next meeting: Richmond, Va., October, 1932.

Cuba

Sociedad Cubana de Radiologia y Fisioterapia
Secretary, Dr. Luis Fariñas, Animas 110, Havana, Cuba.
Meets monthly in Havana.

British Empire

British Institute of Radiology Incorporated with the Röntgen Society
Meets on the third Thursday of each month, from November to June inclusive, at 8:15 p.m., at 32 Welbeck St., London, W. 1., or as advertised.

Electro-Therapeutic Section of the Royal Society of Medicine (Confined to Medical Members)
Meets on the third Friday of each month during the winter at 8:30 p.m. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1.

Section of Radiology and Medical Electricity, Australasian Medical Congress
Secretary, Dr. H. M. Cutler, 139 Macquarie St., Sydney, New South Wales.

Radiological Section of the Victorian Branch of the British Medical Association
Secretary, Dr. Colin Macdonald, Lister House, 61 Collins St., Melbourne, Australia.
Meets monthly at Melbourne during the winter.

Section on Radiology, Canadian Medical Association
Secretary, Dr. A. H. Rolph, 160 St. George St., Toronto, Ont.

Radiological Section, New Zealand British Medical Association
Secretary, Dr. P. C. Fenwick, The Hospital, Christchurch. Meets annually.

Continental Europe

Belgian Society of Roentgenology
Secretary, Dr. J. Boine, Avenue des Allies, 134, Louvain, (Belgium).
Meets monthly on second Sunday at d'Egmonds Palace, Brussels, except in the summertime.

Societe de Radiologie Médicale de France
Meets monthly on second Tuesday, except during months of August and September, 12 Rue de Seine, Paris.

Société Suisse de Radiologie (Schweizerische Röntgen-Gesellschaft)
Secretary for French language, Dr. A. Grosjean, La Chaux de Fonds.
Secretary for German language, Dr. Scheurer, Molzgasse, Biel.
Meets annually in different cities.

Société Francaise d'Electrothérapie et de Radiologie Médicale
Meets monthly on fourth Tuesday, except during months of August and September, 12 Rue de Seine, Paris.

Association of German Roentgenologists and Radiologists in Czecho-Slovakia
Secretary, Dr. Walter Altschul, German University, Prague, 11/52.

Deutsche Röntgen-Gesellschaft (Gesellschaft für Röntgenkunde und Strahlenforschung)
Meets annually in April, alternating one year in Berlin, one year in some other German city. Meets in addition every two years with the Gesellschaft deutscher Naturforscher und Aerzte.
Permanent secretary, Professor Dr. Haenisch, Klopstock strasse 10, Hamburg, Germany.

Süd-und Westdeutsche Röntgengesellschaft
Meets annually in different cities.

Nord- und Ostdeutsche Roentgengesellschaft
Meets annually in different cities.

Dutch Society of Electrology and Roentgenology
Holds two meetings a year in Amsterdam, one in the Spring, and one in the Fall.

Societa Italiana Radiologia Medica
Secretary, Professor M. Ponzio, University of Turin, Turin.

Societatea Romana de Radiologie si Electrologie
Secretary, Dr. Oscar Meller, Str. Banul Maracine 30, Bucarest, Rumania.
Meets second Monday in every month with the exception of July and August.

All-Russian Roentgen Ray Association, Leningrad, USSR in the State Institute of Roentgenology and Radiology, 6 Roentgen St.
Secretaries, Drs. S. A. Reinberg and S. G. Simonson.
Meets annually.

Leningrad Roentgen Ray Society
Secretaries, Drs. S. G. Simonson and G. A. Gusterin.
Meets monthly, first Monday at 8 o'clock, State Institute of Roentgenology and Radiology, Leningrad.

Moscow Roentgen Ray Society
Secretaries, Drs. L. L. Holst, A. W. Ssamygin and S. T. Konobejevsky.
Meets monthly on the first Monday at 8 o'clock, the place of meeting being selected by the Society.

Polish Society of Radiology
Secretary, Dr. Jan. Kochanowski, 45 Gornoslazka St., Warsaw. Meets annually.

Warsaw Section, Polish Society of Radiology
Secretary, Dr. B. Krynski, 11 Zielna St.
Meets once a month except in the summertime.

Scandinavian Roentgen Societies
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association. Each of the following societies, with exception of the Denmark Society, meets every second month except in the summertime.

Society of Medical Radiology of Sweden
Meets in Stockholm.

Society of Medical Radiology in Norway
Meets in Oslo.

Society of Medical Radiology in Denmark
Secretary, Dr. G. Biering, Copenhagen.
Meets on the second Wednesday of each month from October to July in Copenhagen, at 8 o'clock in the State Institute of Roentgenology.

Society of Medical Radiology in Finland
Meets in Helsingfors.

Vienna Society of Roentgenology
Meets first Tuesday each month, October to July.

Orient

Japan X-ray Association
c/o Orthopedic Surgery, Tokyo Imperial University.
Meets annually in April.

Kinki Roentgen-Abend Society
Director, Dr. Prof. Taiga Saito, Ogawaoike, Kyoto, Japan. Meets bi-monthly on third Sunday.

THE SCIENTIFIC EXHIBIT

The scientific exhibits for the Thirty-third Annual Meeting of the Roentgen Ray Society were assembled and displayed under the direction of Dr. S. W. Donaldson. To him and to the various members of the Society and their guests who contributed to this exhibit the Society desires to express its thanks.

The exhibits included the following: A demonstration of Injuries of the Wrist and Kienböck's Disease by Drs. A. W. Erskine and Carl L. Gillies, Cedar Rapids, Iowa. The various films in this exhibit demonstrated the common fractures of the wrist in children and adults, and also there were films demonstrating the rare and unusual fractures and dislocations of the carpal bones, including four cases of Kienböck's disease.

An exhibit on Tuberculosis of Bones and Joints was arranged by Dr. Maurice M. Pomeranz, New York City. This exhibit included films showing the development of tuberculous lesions, also films of specific cases, particularly those in which the diagnosis at the beginning was doubtful, and a number of films showing an unusual manifestation of the process, as well as those showing the typical manner in which tuberculosis either destroys bone or heals by non-operative measures.

Dr. Charles L. Martin and Dr. J. M. Martin of Dallas, Texas, had a splendid exhibit illustrating the Small Radium Needle Technique in the Treatment of Cancer.

Dr. C. B. Peirce, Ann Arbor, Mich., had an excellent exhibit on the superior mediastinum. The films illustrated the importance of the lateral view in the study of the mediastinum, and the anatomic relations of this portion of the thorax were shown with roentgen studies of the normal and abnormal cases. Dr. Peirce also had an exhibit on Giant Cell Bone Tumor, varieties of which were shown in photomicrographic studies demonstrating the reaction after roentgen therapy.

Dr. J. Thompson Stevens, Montclair, N. J., had an excellent exhibit on the Results of the Treatment of Accessible Malignancies with Radium, Roentgen Rays and Electrothermic Surgery. This consisted of a number of photographs of patients before and after treatment for malignant disease of the skin, mouth, breast and lip.

Drs. S. W. Donaldson, A. C. Furstenburg, and C. B. Peirce, Ann Arbor, Mich., had an exhibit on Osteomyelitis of the Skull, which consisted of a number of reproductions showing osteomyelitis of the skull in the various preoperative lesions, the postoperative defect and evidence of bone repair. These films illustrated that in some cases the spread of infection is by continuity of tissue; in others, the infection spreads through the intracranial diploic veins. Various films were shown in this exhibit demonstrating the osteogenic processes in the repair of cranial defects, illustrating how the outer layer of the dura, virtually a periosteal element, passes through typical phases of bone growth.

Dr. G. E. Richards, Toronto, Ont., had an excellent exhibit on The Interpretation of Triangular Basal Shadows. This exhibit served to illustrate the paper given by Dr. Richards on this subject. It was one of the most instructive and comprehensive exhibits shown at the meeting. Dr. Richards also had an exhibit on the Treatment of External Cancer by X-rays and Radium. This consisted of photographs in the form of transparencies illustrating the results of treatment in a variety of external lesions.

Dr. H. K. Taylor, New York City, had a splendid exhibit on the Roentgen Findings in Suppuration of the Petrous Apex. This served to illustrate the various type cases which Dr. Taylor discussed in his paper on this subject. Both the paper and the exhibit were of enormous clinical importance.

Dr. J. W. Towey and Dr. John K. Parish, Powers, Mich., had an exhibit on X-ray Findings Produced by Fungous Spores. This consisted of a roentgenologic study made on a group of patients all of whom were employed in the manufacture of wood products and who had become affected

by the fungous spores found in maple bark. The predominating symptom is shortness of breath and this is associated with cough and loss of weight. The patients improve as soon as they are removed from the environment of the spores and the roentgenograms show relatively rapid clearing of the inflammatory process that develops in the lungs.

Dr. F. F. Borzell, Philadelphia, had a series of films showing the bone changes observed in three cases of erythroblastic anemia. These illustrated Dr. Borzell's discussion of Cooley's syndrome.

Dr. Albert A. Rayle, Atlanta, Ga., had an exhibit on Tumors of Bone. This exhibit included a selection from the large number of osseous tumors which have been seen at the Steiner Clinic during the past seven years. The exhibit was divided into: (*a*) primary malignant tumors: (*b*) secondary malignancies; (*c*) a few non-tumor cases where differentiation from tumor is more or less difficult.

Drs. John Alexander, Cameron Haight and Leonard Burr, Ann Arbor, Mich., had a splendid exhibit illustrating the Surgical Measures in the Treatment of Pulmonary Tuberculosis. This was a demonstration of the indications for and results of surgical measures used for pulmonary tuberculosis by means of roentgenograms, drawings, and photographs: Temporary and permanent complete phrenic nerve interruption; closed and open intrapleural pneumolysis; extrapleural pneumolysis with paraffin, pectoral muscles and rubber bag fillings; scaleniectomy; multiple temporary and permanent intercostal neurectomy; paravertebral and anterolateral thoracoplasty.

Drs. E. E. Downs and W. S. Hastings, Philadelphia, had an exhibit on Bone Metastases. Twenty cases of bone metastases were shown together with the color photomicrographs of the primary tumors. This exhibit illustrated their paper on "Factors Influencing the Type of Metastatic Carcinoma of Bone" in which evidence was offered of a degree of parallelism between the histological characteristics of the primary tumors and the type of bone metastases found. They pointed out the importance in their paper and illustrated in this exhibit that too little attention has previously been given in the literature to the individual growth habits of the tumor in considering the question of the production of osteolytic or osteoplastic metastatic lesions.

Dr. Ralph S. Bromer, Philadelphia, had an exhibit on Osteogenesis Imperfecta.

Drs. L. S. Otell, A. C. Christie, E. A. Merritt, and T. A. Groover, Washington, D. C., had a splendid exhibit on Hepatosplenography.

Dr. I. H. Lockwood, Kansas City, Mo., had an exhibit on the Roentgen Examination of the Breast. This exhibit served to illustrate and enhance the value of Dr. Lockwood's paper on "The Roentgen-Ray Evaluation of Breast Symptoms." The exhibit included the technique of examination, the classification of the various tumors and the various phases which the breast undergoes during the menstrual cycle, and the exhibit served to emphasize the importance of the roentgen examination as an accurate supplementary diagnostic method for the early recognition of cancer as well as other diseases of the breast.

The Detroit Board of Health had a splendid exhibit on the Pulmonary Cavity and its Control. This exhibit illustrated the types of cavities, the various lesions amenable to collapse therapy and the results of the treatment of a variety of cavities by the several surgical procedures.

Dr. R. S. Rowland, Detroit, had an exhibit on Schüller-Christian's Disease.

Drs. Max Ballin and Plinn F. Morse, Detroit, had a splendid exhibit on Parathyroidism—Its Symptoms and Surgery. There were plates showing the following: clinically observed progressed cases of parathyroidism, with the roentgenology and pathology; operated cases of parathyroid tumors showing their bony changes, notes on giant cell tumors, cysts, etc., and results of operation; affections of jaw due to parathyroidism, arthritic type; parathyroidism, Paget type; some outstanding cases from

the literature; differential diagnosis, osteopoicilia, myeloma, thymoma and metastatic cancer; composite X-rays of the whole skeleton of the juvenile type; skull changes in parathyroidism; spinal changes in parathyroidism; parathyroid changes in the pelvis and hips; changes in extremities; surgical anatomy of the parathyroids; results of parathyroidectomy in Paget's disease; other endocrine changes in bones; the giant cell tumor in parathyroidism.

Drs. J. G. Mateer, H. P. Doub and F. W. Hartman, Detroit, had an exhibit on Carcinoma of the Duodenum in which a description was given of a study of six cases of carcinoma of the duodenum. The exhibit included charts showing the clinical findings in the supra-ampullary, ampullary and infra-ampullary regions, and films illustrating the roentgen findings as well as the autopsy findings with microscopical studies in each type of case.

A new feature of the scientific exhibit was an Industrial Exhibit which was participated in by the following: Mr. A. J. Moses, Combustion Engineering Co., Hedges, Walsh and Weidner Plant, Chattanooga, Tenn., under the head of Welding and Annealing gave an excellent exhibit of a complete collection of films showing the work in different phases of welding and annealing of different metals and the advances which have been made in different processes since roentgen inspection was inaugurated.

Mr. D. W. Saxton, Hoover Steel Ball Co., Ann Arbor, Mich., had an exhibit on Grinding Wheels. Six wheels were shown with defects. These included cracks while being burned in the kiln and wheels which were cracked while being shaved green before burning. There were also shown roentgenograms of wheels that were uneven in density or bond structure and some uneven in grade, meaning soft in some parts and hard in others. Eight wheels were made expressly for roentgen-ray experimentation. Two wheels of the same dimensions and base as shellac, rubber, bakelite, etc., were made, one being perfect and the other purposely of unsatisfactory structure. Roentgenograms were made of each and were so mounted as to allow comparison and inspection.

The exhibit of Prof. Lars Thomassen, Department of Engineering, University of Michigan, on Changes in Metal Structure, consisted in roentgenograms showing the effects of cold working (hammering) on brass; also films showing the changes in structure after cold working on steel and after annealing of steel. Included in the exhibit were roentgenographic demonstrations of defects in high pressure steam valves.

Mr. C. A. Poole, Director Industrial Research, Kelley-Koett Mfg. Co., Covington, Ky., had an exhibit on Applications of X-ray in the Non-medical Field. This showed the possibilities of demonstrating hidden defects in metal products which would escape ordinary factory inspection. Many other uses of the roentgen ray in the commercial field were included and films were so arranged that they could be viewed without breaking the continuity of those made in series.

The X-ray Examination of Welded Boiler Plate by Mr. R. R. Kondal, Engineering Department, Wickes Boiler Co., Saginaw, Mich., consisted of (1) film showing 1-inch thick boiler plate welded together with a perfect weld but with a difference in density due to rust or scale on the plate; (2) cracks or laminations within a steel pipe and with porosity at the welded end; (3) a 3-inch steel plate welded together and with demonstrable slag inclusions to be chipped out and rewelded; (4) numerous gas pockets in a very porous weld within a $1\frac{1}{2}$-inch thick steel plate.

The Historical Exhibit consisted of photographs of all the past presidents of the American Roentgen Ray Society, a complete exhibit of tubes used at Eloise Hospital, with equipment used in obtaining the first roentgenogram made at that hospital in 1895.

There was an excellent exhibit from the Section on Roentgenology of the Mayo Clinic, Rochester, Minn., by Drs. B. R.

Kirklin, John D. Camp and H. M. Weber, which included (1) cholecystograms, drawings and photographs illustrating the technique of examination and the diagnosis of tumors of the gallbladder; (2) roentgenograms of surgically proved cases of hypertrophy of the pyloric muscle; (3) roentgenograms depicting the various bone changes found in cases of hyperparathyroidism. The progression of these changes during the disease and their regression following the removal of parathyroid tumors were shown, and the characteristic roentgenographic phenomena which permit a diagnosis of hyperparathyroidism were illustrated; (4) an exhibit illustrating the roentgenographic manifestations of the more commonly encountered lesions of the colon. This consisted of two units, one dealing with the neoplastic lesions of the large intestine, and the other with the non-neoplastic diseases. The specimens were selected especially with the illustration of anatomic differential diagnosis in mind. Because it is considered of particular value for the purpose, the "double contrast method" found a prominent place in the exhibit.

There was a demonstration entitled Reading with Closed Eyes, given by Dr. A. H. Pirie, Montreal. His description of this demonstration is as follows: The retina becomes very sensitive when kept in the dark for a long time. When a powerful beam of light falls on the retina, a bright flash of light is seen. Whether the eyes are closed or not makes very little difference. Lead letters placed in the path of the rays cast their shadows on the retina. The patient sees the letters upside down and turned from right to left. A patient blind from destruction to the front part of the eye can thus see the letters that the ray has projected on the retina as described, from lead letters. Applications and uses of the ray—with eyes shut and using no screen. (1) A foreign body in the eye can be made to cast its shadow and thus help in the localization of it. (2) The patient's localization is exactly the reverse of the roentgenologist's. (3) Detachment of the retina and tears in the retina due to a foreign body can be located by the patient. (4) The field of vision can be mapped out by passing a slit of roentgen rays across the eyes in two directions; or more simply viewing brass mosquito netting specially marked with a cross and circles. (5) In case of complete cataract, the condition of the retina can be investigated: (*a*) small pictures painted with lead paint; (*b*) seeing a small photograph about 1 cm. square taken on special thick films very rich in silver; (*c*) viewing two stereoscopic films and seeing them stereoscopically; (*d*) peculiar effects caused by shadows projected in straight lines on the cup of the retina.

The Committee on Awards for the Scientific Exhibit composed of Dr. Ross Golden, Chairman, Dr. Vincent W. Archer and H. J. Walton, made the following report:

The committee appointed to judge the scientific exhibits was instructed to recommend some form of permanent memento to be presented in the name of the Society to those receiving first, second and third place. Following a suggestion made at the last executive session, we recommend, therefore, that these mementos be gold, silver and bronze medals having on one side the official seal of the Society and on the other the facts of the presentation engraved in words to be chosen by the secretary. We recommend that the secretary be empowered to order these medals, to select their size and to decide upon other details after consultation with the engravers.

The committee has examined the scientific exhibits with unqualified approval. As a whole, they certainly equal if not exceed in interest and educational value those presented at previous meetings. The committee strongly urges those who have not examined them to do so.

We have found it difficult to make our decisions and have attempted to base a judgment on four points: (1) originality; (2) clinical importance; (3) educational value, and (4) method or presentation.

We recommend, therefore, the following awards:

1. Gold medal to exhibit No. 24: Demonstration—Reading with Closed Eyes, by Dr. A. H. Pirie of Montreal.

2. Silver medal to exhibit No. 12: Surgical measures in the Treatment of Pulmonary Tuberculosis, by Drs. John Alexander, Cameron Haight, and Leonard Burr, of Ann Arbor, Michigan.

3. Bronze medal to exhibit No. 19: Parathyroidism—Its Symptoms and Surgery, by Drs. Max Ballin and Plinn F. Morse, Detroit, Michigan.

4. Honorable Mention to exhibit No. 23: Exhibit from the Section on Roentgenology, Mayo Clinic, by Drs. B. R. Kirklin, John D. Camp and Harry M. Weber, with special approval of Dr. Weber's work on the colon; to exhibit No. 3: Small Radium Needle Technique in the Treatment of Cancer, by Drs. Charles L. Martin and J. M. Martin, Dallas, Texas; and to exhibit No. 4: The Superior Mediastinum, by Dr. Carleton B. Peirce, Ann Arbor, Michigan.

Special mention should be made of the exhibit on the industrial applications of the roentgen ray.

THE COMMERCIAL EXHIBIT

The American Roentgen Ray Society desires to express its sincere thanks and appreciation to the following companies who had exhibits at the Detroit meeting: George W. Brady and Company, Buck X-Ograph Company, Eastman Kodak Company, General Electric X-Ray Corporation, Kelley-Koett Manufacturing Company, Metalix Service Company, Patterson Screen Company, Picker X-Ray Corporation, Standard X-Ray Company, Charles C Thomas, Publisher, Victoreen Instrument Company, Westinghouse X-Ray Company.

The various exhibits included the commoner paraphernalia and equipment which are ordinarily used in the roentgen laboratory but in addition many new features which included not only changes in models of the ordinary roentgen-ray tables and accessories used but also changes in construction of the generators, particularly the therapy generators, as well as the shockproof units for head roentgenography and the roentgenographic display of films made at high milliamperage. The members of the Society and guests who availed themselves of the opportunity to visit the commercial exhibit were fully recompensed for the time spent there as the exhibitors were always present to explain their equipment and it is of course always a source of pleasure to meet the various representatives and manufacturers who have themselves contributed so much to the advancement of roentgenology.

AMERICAN ASSOCIATION FOR THE ADVANCEMENT OF SCIENCE

The winter meeting of the American Association for the Advancement of Science and associated societies will be held in Atlantic City, N. J., December 27 to 31, 1932. Particular attention is called to the program of Section N which should appeal especially to the medical profession. The Section N program will extend over two days, Wednesday and Thursday, December 28 and 29. It consists of the retiring vice-presidential address by Dr. Howard T. Karsner on "Medieval Guilds of Medical Interest"; a symposium on the "Physiological Relations of the Pituitary Body" which is in charge of Dr. J. J. Abel, the President of the Association; and a symposium on "Filterable Viruses and Filterable Virus Diseases" which is in charge of Dr. William H. Park, the Vice-President for Section N.

The symposium on the pituitary body promises to be of very unusual interest. Dr. Abel himself will discuss the relations of the posterior lobe and Dr. Phillip E. Smith, Dr. E. T. Engle and Dr. Herbert Evans will discuss the anterior lobe.

In the symposium on filterable viruses and filterable virus diseases, Dr. William H. Park will speak on poliomyelitis. Dr. W. A. Sawyer will give the recent work on yellow fever, and other phases of the subject will be discussed by Dr. E. V. Cowdry, Dr. Hans Zinsser, Dr. R. R. Hyde, Dr. E. B. McKinley and Dr. T. M. Rivers. Owing to recent discussions on the relation of bacteria and filterable viruses this symposium is of timely interest.

Further information may be obtained by writing the Permanent Secretary of the

Association, Dr. Charles F. Roos, Smithsonian Institution Building, Washington, D. C.

SOUTH CAROLINA X-RAY SOCIETY

On June 9, 1932, at a meeting held in the Medical Building, Columbia, the roentgenologists of the state of South Carolina formed the South Carolina X-Ray Society. The following were made charter members: Dr. F. D. Rodgers, Columbia; Dr. R. B. Taft, Charleston; Dr. P. D. Hay, Florence; Dr. O. D. Baxter, Sumter; Dr. Hillyer Rudisill, Charleston; Dr. W. S. Judy, Greenville; Dr. T. A. Pitts, Columbia; Dr. W. M. Sheridan, Spartansburg; Dr. F. R. Wrenn, Anderson, and Dr. Malcolm Mosteller, Columbia. Dr. F. D. Rodgers of Columbia was elected President and Dr. R. B. Taft of Charleston was elected Secretary. Meetings will be held at the time and place of the South Carolina State Medical Association meeting. The next meeting will be in Spartansburg, April, 1933.

BOOK REVIEWS

Books Received Are Acknowledged under Heading: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers as space permits.

Intracranial Tumours: Notes upon a Series of Two Thousand Verified Cases with Surgical-Mortality Percentages Pertaining Thereto. By Harvey Cushing, Professor of Surgery, Harvard Medical School; Surgeon-in-Chief, Peter Bent Brigham Hospital, Boston. Cloth. Price, $5.00. Pp. 150, with 111 illustrations. Springfield, Illinois; Baltimore, Maryland: Charles C Thomas, 1932.

This monograph formed the basis of a report made before the International Neurological Congress at Berne. It contains the essence of thirty full years of experience with the intracranial tumors. Each of the twelve short chapters provides fascinating reading in spite of the fact that it deals mainly with statistics of surgical mortality in the respective tumor groups. If, as the author states, this is the last report he shall ever attempt to make on the subject of brain tumors as a whole, he is surely hanging up the great bow of Ulysses to test the strength of the neurological surgeons of the future. The low surgical mortality in this unprecedented group of over two thousand verified cases sets up a most difficult mark to shoot at.

The first four chapters deal with the four largest intracranial tumor groups, namely, gliomas, pituitary adenomas, meningiomas, and acoustic tumors. The remaining chapters are devoted to the rarer tumors.

I. The gliomas, of which there are 862 in the series, vary greatly in malignancy according to their histological classification. Special reference is made to the supravital staining methods as developed by Dr. Louise Eisenhardt in the laboratory of the author. Bits of tumor tissue removed at operation can be immediately diagnosed by this method, making it possible to deal more properly with the exposed tumor.

Astrocytomas of the cerebellum, as a group, are the most favorable gliomas from the standpoint of survival period. Their operative mortality for the last three years is 2.9 per cent.

The 208 glioblastomas (also called spongioblastoma multiforme) are highly malignant with an average postoperative span of only six to nine months before recurrence of symptoms. A case treated with intensive irradiation is cited in this group with a symptom-free interval of three years, yet the author in his comments belittles the effect of radiation in the glioblastomas as a whole.

The medulloblastomas are also highly malignant gliomas which frequently metastasize to the spinal canal. Of all the intracranial tumors, the author believes these to be the most radiosensitive. However, radiation should be given only after operation and verification of the tissue type.

The rarer oligodendrogliomas and ependymomas among the gliomas are frequently diagnosed from their spotty calcification as shown in the roentgenogram.

The figures for the past three years give a 15.7 per cent case and a 11.0 per cent operative mortality for gliomas as a whole, irrespective of their type of position.

II. Pituitary adenomas form the next large group of 360 verified tumors. The advantages of the transfrontal over the earlier transphenoidal approach are discussed. The author finds that the chromophile adenomas are somewhat susceptible to radiation but he believes that radiotherapeutics of the chromophobe adenomas, at least, will be discontinued as soon as the neurosurgeons as a class perfect themselves in the details of operative procedure. The operative mortality of the whole group is 6.2 per cent and the case mortality, 7.1 per cent.

III. The slow-growing meningiomas may form huge tumors and although technically they offer more difficulty than any other group, they are the most favorable of the intracranial tumors for surgical attack. These tumors have their favored sites within the cranial cavity and a corresponding symptomatology. Even in the absence of roentgenological indications of their precise point of origin as betrayed by cranial abnormalities, the several syndromes according to situation tend to become increasingly clear. Of the 271 verified meningiomas, 260 were operated on, with an operative mortality of 11 per cent.

IV. The acoustic tumor statistics have greatly improved in recent years, due mainly to (1) the use of local anesthesia; (2) employment

of suction to supplement the intracapsular spooning out of the lesion; (3) electrosurgery; (4) uncapping the cerebellum to expose the tumor. It is pointed out that after the cerebellum is uncapped by electrosurgical means, there is no increased ataxia if the dentate nucleus is not disturbed, and the improved exposure tends to lower the mortality rate. Of 171 acoustic tumors operated on, the total operative mortality was 11.4 per cent, while the mortality for the last three-year period was only 3.4 per cent.

The remaining chapters take up congenital, granulomatous, metastatic and vascular intracranial tumors. With the vascular malformations, the author believes that radiation should be used rather than any attempt being made to excise them.

The final chapter sums up the operative statistics. A comparison is made between the mortality rates at various periods in the author's experience. The operative mortality of the four major groups of verified tumors during the last three-year period is one-third to one-fifth of what it was in the series before 1912. The author attributes this improvement to better surgical technique and more expert postoperative nursing rather than to earlier diagnosis, and concludes by paying tribute to his successive assistants and coworkers.

Frederic Schreiber

Röntgendiagnostik des Schädels bei Erkrankungen des Auges und seiner Nachbarorgane. (Roentgen Diagnosis of the Skull with Reference to the Eyes and Neighboring Organs.) Von Dr. Rudolf Thiel, a. o. Professor der Augenheilkunde an der Universität Berlin, Assistent der Universitäts-Augenklinik, Berlin. Paper, RM. 48.00; bound, RM. 54.00. Pp. 87, with 411 illustrations. Berlin: Julius Springer, 1932.

Dr. Thiel has produced a book which is an excellent example of painstaking detail and which covers the subject completely. Each disease of the eye which is manifested in such a way as to give roentgen signs within the orbit or by involvement of the neighboring tissues is presented with illustrative cases. Developmental deformities, osteodystrophies, tumors, and traumatic conditions of the orbit and intraorbital structures are first discussed. Inflammatory processes, tumors and injuries of the paranasal sinuses are next considered, and chapters follow dealing with the middle fossa of the skull, especially the sella, diseases showing generalized increase of intracranial pressure, dental conditions affecting the eye, and lastly the lacrymal ducts.

Case presentations include a brief synopsis of history, clinical and laboratory findings, photographs of the patient, visual fields, reproductions of retinoscopic views, roentgenograms, diagrams and photographs of pathologic specimens.

Ventriculograms and lipiodol injections are included. An interesting method of localizing intraocular foreign bodies is described. The discussion of technique and normal anatomy is quite sufficient for the purposes of the work.

The book is published in two parts, the text being separate from the illustrations, but they cannot be bought separately.

The book is highly recommended to roentgenologists, ophthalmalogists and all others interested in the subject.

E. W. Hall

Fungous Diseases: A Clinico-Mycological Text. By Harry P. Jacobson, M.D. Attending Dermatologist and Member of the Malignancy Board, Los Angeles County General Hospital. With introductions by Jay Frank Schamberg, M.D., Professor of Dermatology and Syphilology, Graduate School of Medicine, University of Pennsylvania, and Howard Morrow, M.D., Clinical Professor of Dermatology, University of California Medical School. Cloth. Price, $5.50. Pp. 317, with 153 illustrations. Springfield, Illinois: Charles C Thomas, 1932.

The radiologist, regardless of whether his interest is chiefly diagnostic or therapeutic, has frequent need of a well-rounded knowledge of the fungous diseases. Much has been learned about these conditions during the past decade but the information is scattered and often incomplete. Dr. Jacobson has gathered the best of the literature and by adding the fruits of his own experience has compiled a short, readable monograph which makes the available knowledge readily accessible.

The first sixty-three pages of the book deal with primary cutaneous mycoses that usually show no systemic involvement. Under this heading are considered dermatoses produced by microspora, trichophyta, epidermophyta and achoria. Considerable space is devoted to the laboratory methods used in making the diagnoses and the clinical manifestations are well described and illustrated with many photo-

graphs. The usual therapeutic procedures including roentgen-ray epilation for ringworm of the scalp and bearded regions are advocated. Radiologists will no doubt read with some surprise the author's advice to the effect that the rather dangerous drug, thallium acetate, should be given along with the roentgen exposure "to lessen the quantity of x-rays needed for epilation." It is the reviewer's impression that no proof of any such effect on the epilation dose is available and that a properly measured dose of roentgen rays given alone is a safer procedure.

The remainder of the book deals with infections of the skin and mucous membranes which are frequently accompanied by systemic involvement. The conditions considered are moniliasis, mycetoma, sporotrichosis, blastomycosis, actinomycosis, coccidioides, torulosis, and aspergillosis. Some of these diseases produce lesions in the lungs, bones, cerebrospinal system, intestinal tract and genitourinary system which closely resemble those seen with syphilis and tuberculosis. Even malignancy enters into the differential diagnosis at times. Roentgenologists should be particularly interested in the pulmonary and osseous manifestations of the fungous diseases as they are seen roentgenographically and unfortunately this phase of the subject is very briefly dealt with. Under the heading of Therapy, roentgen treatment of deep-seated lesions is mentioned frequently but no exact technique is given and one is led to believe that the author is not intimately acquainted with the method. The section on Coccidioides is the outstanding feature of the book because it covers a wealth of first-hand knowledge which is not so evident in some of the other sections.

Although this book deals for the most part with the laboratory and clinical aspects of mycotic infections, the radiologist will find it a well-written and accurate reference book filled with valuable information concerning his occasional atypical case. If he happens to reside on the Pacific Coast where fungous infections are relatively common, his need for the text will be even greater.

Charles L. Martin

DEPARTMENT OF TECHNIQUE

Department Editor: Robert B. Taft, 105 Rutledge Ave., Charleston, S. C.

A SIMPLE QUICK CENTERING APPARATUS FOR STEREOSCOPIC MASTOID ROENTGENOGRAPHY

By BORGE W. PETERSEN
X-ray Clinic of Cornell University Medical College
NEW YORK CITY

THERE is a need of a mastoid block which permits rapid centering and which can be constructed at slight expense from parts usually found in a general roentgen laboratory. The following apparatus, while incorporating no startling original features, has been found to be of considerable help in obtaining good mastoid films. As can be seen on the illustrations, the block consists of 12° inclined top, underneath which has been placed a mirror for centering. The top is made of brass in which two circles (4″ in diameter) have been cut for exposures. These openings are covered with transparent celluloid on which the centers are indicated. In Figure 1 the openings and their center marks are seen reflected in the mirror.

Fig. 1. Patient in position. Note the plain visible outline of forward bended soft tissue of ear. In this case the mark indicating the external auditory channel coincides with the center mark on the apparatus.

Fig. 2. Cassette in groove. Note the position of technician.

In posturing the patient the area it is desired to center is marked on the patient with a skin-pencil. Using the mirror as a guide this is made to coincide with the center mark of the opening. After the patient is postured, the cassette is placed in a

groove close up under the inclined top and the exposure is made.

Stereoscopic films are obtained by using two cassettes. After the first exposure has been made on cassette I, the second of the stereoscopic pair is made on cassette II, marking the cassettes so they will be placed properly for exposures of the other ear. The cassettes must be marked I and II, and the letter *L* to indicate upper left.

A similar arrangement is suggested for stereoscopic sinus work.

PROTECTIVE COVER FOR TUBE BOWL

By ROBERT B. TAFT, M.D., B.S.
CHARLESTON, SOUTH CAROLINA

THE amount of stray radiation emerging from the open top of the conventional type of lead glass tube bowl is quite significant and becomes really dangerous

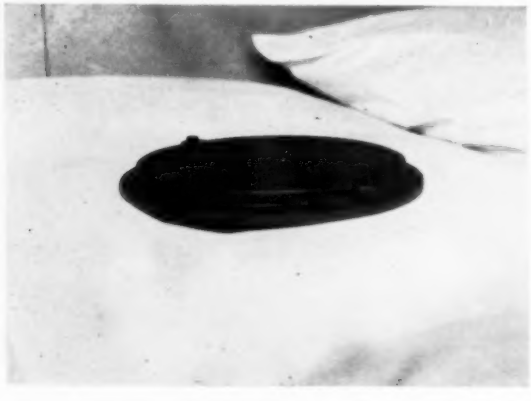


FIG. 1.

if persons are working all day on the floor above the roentgenographic or therapeutic rooms.

An extremely simple device to prevent this radiation is shown in Figures 1 and 2. The protective material is lead rubber of $\frac{1}{8}$ inch thickness between two circular pieces of thin plywood. One of these disks is of such a size that it fits snugly into the top of the bowl and the other is of the same diameter as the outside of the bowl. The circle of lead rubber is glued between the two boards and the two bolted together with bakelite bolts.

No appreciable heating of the tube has been noticed as the result of being more completely enclosed.

In addition to fulfilling its purpose, this cover prevents the tube becoming dusty as rapidly.

As this entire device is made of non-conductive materials, it presents no electrical danger of sparks from the wires.

The disks of plywood can be made in a few minutes in any woodworking shop. No dimensions are given as the size of different tube bowls varies slightly.

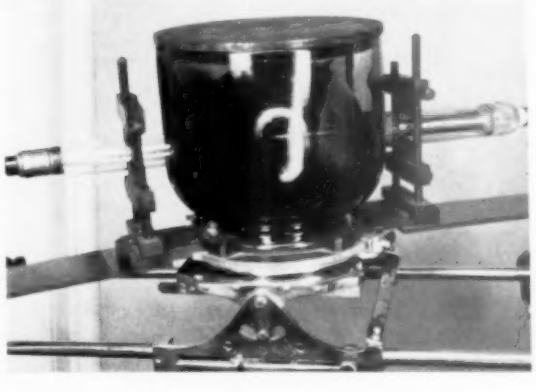


FIG. 2.

INDEX TO ABSTRACTS

ROENTGEN DIAGNOSIS

Head

Laruelle, L.: Estimation of the cerebral ventricles by a routine procedure..... 563

Jouret, Jos.: Cerebral pneumoventriculography; procedure of ventricular estimation according to Dr. Laruelle..... 564

Giradui, Gioffredo: The syndrome of the tumors of the apex of the temporal pyramid..... 564

Payne, Reginald T.: Sialography..... 565

Neck and Chest

Pape, R., and Spitznagel, K.: Myomas of the esophagus..... 565

Jacobi, Mendel, and Rascoff, Henry: Congenital esophageal stenosis associated with aberrant ductus arteriosus..... 565

Uspensky, A., and Helfon, A.: The significance of the roentgen ray for the study of bronchiectasis..... 566

Podkaminsky, N. A.: Roentgenologic aspect of pulmonary siderosis..... 566

ABSTRACTS OF ROENTGEN AND RADIUM LITERATURE

ROENTGEN DIAGNOSIS

HEAD

Laruelle, L. Le repérage des ventricules cérébraux par un procédé de routine. (Estimation of the cerebral ventricles by a routine procedure.) *Presse méd.*, Dec. 23, 1931, *39*, 1888–1891.

The procedure called "ventricular estimation" differs from the ventriculography of Dandy in the following points: (1) its object is to verify the accessibility of the cerebral ventricles to the air injected by the spinal route, the method forming in this respect a veritable "test of circulation," and to demonstrate the anomalies in form, size, contour, etc., of the cerebral ventricles; (2) it uses exclusively the spinal route, and (3) it is performed with the least amount of air possible.

The technique is very simple. Ten cubic centimeters (in unusual cases 5 or 15 c.c.) of air are injected by lumbar or more rarely by suboccipital puncture, after withdrawal of a larger amount of cerebrospinal fluid. The procedure is carried out with the patient in sitting position. After completion of the injection, the head is slightly flexed so as to facilitate the passage of the air through the posterior arachnoid space at the level of the occipital foramen. Stereoscopic roentgenograms are made in both the lateral and anteroposterior views and the relative position of the air bubbles in the two ventricles estimated. In certain instances it is advisable to test also the lateral circulation through the third ventricle by placing the patient alternately on the right and left side for fifteen minutes.

There are two inconveniences to the method: (1) the air may take a false root and thus not reach the cerebral ventricles, and (2) the two ventricles may not absorb the same quantity of air, this resulting in unequal images. These may be obviated by repeating the examination two or three times.

The procedure was used in 200 cases, 20 of which proved to be cerebral tumors and were controlled either by operation or by autopsy. It was found that the accuracy of the diagnosis in the 20 cases was 94 per cent, which coincides with the figures of the usual ventriculography.

The great advantage of the method lies in the fact that it is exceedingly easy to carry out, the mortality is nil and the subjective reaction of the patients negligible. Of course, it does not give a comprehensive roentgen study of the entire ventricular system but if such is required the "estimation" can always be supplemented by encephalography or ventriculography accord-

ing to Dandy and by arterial encephalography according to Egas Moniz.—*T. Leucutia.*

Jouret, Jos. Pneumoventriculographie cérébrale; procédé de repérage ventriculaire du Dr. Laruelle. (Cerebral pneumoventriculography; procedure of ventricular estimation according to Dr. Laruelle.) *J. de radiol. et d'électrol.*, Sept., 1931, *15*, 516–519.

After briefly discussing the principle of the method as well as the technique of procedure (see above abstract) the author attributes the following advantages to "ventricular estimation" as practised by Laruelle: establishment as to whether or not the ventricles are in direct communication with the subarachnoid space, whether or not they occupy a normal position in relation to the median line and whether or not their upper limit is at the same level and same distance from the cranial vertex; demonstration of partial or total deformities, etc. In case of cerebral tumors, the ventricular estimation permits an exact localization and a more or less precise evaluation of the extension of the tumor. In other lesions as, for example, anterior meningitis, inflammations of the choroid plexus, infections of tuberculous or syphilitic origin, etc., the method gives information concerning the pathologic reactions of the cerebral ventricles. Finally, in Jacksonian epilepsy due to traumatism, it permits the eventual localization of cicatricial tissues.—*T. Leucutia.*

Giraudi, Gioffredo. La sindrome dei tumori dell'apice della piramide temporale. (The syndrome of the tumors of the apex of the temporal pyramid.) *Riv. oto-neuro-oftal.*, 1931, *8*, 1–62.

The author discusses in lengthy detail the syndrome which may be provoked by tumors invading the apex of the pyramidal portion of the temporal bone. He was able to collect 15 cases from the literature, all of which are described in résumé, special consideration being given to the 4 cases of Piazza Missorici. To these he adds his own personal case which was that of a carcinoma at the epipharynx extending into the neighboring tissues and invading the apex of the pyramidal bone, thus producing a typical syndrome. It was generally found that the syndrome is produced (*a*) by tumors of the apex of the pyramidal bone; (*b*) by tumors of the epipharynx; (*c*) by tumors of the posterior cranial fossa, and (*d*) by tumors of the medial cranial fossa.

In analyzing the syndrome as a whole, one may distinguish three, or occasionally four, subsyndromes; (*a*) auricular syndrome; (*b*) trigeminal syndrome; (*c*) abducens syndrome, and (*d*) syndrome of Claude Bernard-Horner.

(1) The auricular syndrome consists subjectively of unilateral slight diminution of hearing, associated later with tinnitus, a sensation of occlusion of the ear and occasionally vertigo, and objectively, of diffuse congestion of the tympanic membrane (at the otoscopic examination) with simple hyperemic meningitis and eventual catarrhal otitis media. This latter is produced, according to Ruttin, either by a venous or lymphatic stasis or by occlusion of the eustachian tube, or finally, by a subvirulent infection of the mucosa of the antrum with sterile exudate.

(2) The trigeminal syndrome may have a manifold appearance, according to which branch of the nerve is chiefly affected. Generally it comprises (*a*) motor alterations, manifesting themselves clinically as deviation of the mandible, elevation of half of the soft palate, flaccidity of the floor of the mouth, etc.; (*b*) sensory alterations, leading to various types of neuralgia as, for example, neuralgia supraorbitalis, neuralgia infraorbitalis, neuralgia mandibularis, etc.; (*c*) alterations of reflectoric activity as, for example, diminished or abolished corneal reflexes; (*d*) trophic disturbances which not infrequently lead to ophthalmic neuroparalytica; (*e*) circulatoric disturbances of the lacrymal, salivary and nasal glands, and (*f*) vasomotor disturbances, appearing in the form of flushing of the face, hyperemia of the conjunctiva, nasal and buccal mucosa, etc.

(3) The abducens syndrome consists chiefly of paresis or more often paralysis of the musculus rectus lateralis.

(4) The Claude Bernard-Horner syndrome, which is observed only occasionally, and which occurred rather typically in the author's case, is the result of paralysis of the cephalic portion of the sympathetic nerve. Clinically it comprises (*a*) a narrowing of the palpebral slit, (*b*) sinking of the eyeball into the orbital cavity and (*c*) a narrowing of the pupil.

If the invasion of the tumor is more extensive, the seventh, second and twelfth cranial nerves may also show signs of involvement.

The roentgen appearance varies according to the site of the tumor and amount of invasion. By taking roentgenograms in various positions one is nearly always able to demonstrate an

erosion or destruction around the apex of the pyramidal bone. The author discusses in great detail the errors which may lead to faulty roentgen interpretation as well as the differential roentgen diagnosis from other syndromes of the skull. Pathologically a differentiation must be made between the syndrome of the apex pyramidis due to tumors, nonspecific infections, tuberculosis, lues, and trauma.—*T. Leucutia.*

PAYNE, REGINALD T. Sialography: its technique and applications. *Brit. J. Surg.*, July, 1931, *19*, 142–148.

Sialography is the roentgenographic demonstration of the ducts of the salivary glands by means of injections of radiopaque substances. The parotid gland and ducts lend themselves admirably to this purpose. Lipidol is the opaque medium of choice. The technique described by the author is simple, the apparatus consisting of a rubber fountain pen bulb attached to a short length of glass tubing, one end of which is drawn out to a fine point about 1 mm. in diameter. From 0.5 to 1 c.c. of lipiodol injected causes pain over the parotid region or swelling of the gland which indicates that sufficient lipiodol has been injected. The roentgenoscope may be used to determine if the distribution is satisfactory, then stereoscopic films are made. These will demonstrate abnormalities, dilatations and obstructions of the parotid ducts. Four cases are described in which the author found this procedure useful.—*F. H. Decker.*

NECK AND CHEST

PAPE, R., and SPITZNAGEL, K. Über Ösophagusmyome. (Myomas of the esophagus.) *Fortschr. a. d. Geb. d. Röntgenstrahlen*, Nov., 1931, *44*, 616–625.

Two unusual cases of esophageal myomas are described in lengthy detail. In the first case, that of a woman aged sixty-four, five types of tumors occurred simultaneously. Two of these were malignant (one a hypernephroma of the kidney removed five years previously and the second a primary carcinoma of the bronchus) and the other three benign (multiple lipomata and fibromata of the skin and multiple myomata of the esophagus). The myomata were localized chiefly to the lower part of the esophagus some reaching into the stomach (as pedunculated polyps). The findings were confirmed at autopsy.

In the second case, that of a man aged sixty-eight, a single myoma was located to the upper portion of the esophagus, reaching nearly the size of a fist. At the roentgen examination, as well as at operation, it was confused with substernal thyroid and only at autopsy could the correct diagnosis be made.

Two deductions can be made from the study of the above cases: first, that in an atypical filling of the esophagus one should always bear in mind the possibility of myoma, and second, that a barium examination of the esophagus is indicated in every case of struma.—*T. Leucutia.*

JACOBI, MENDEL, and RASCOFF, HENRY. Congenital esophageal stenosis associated with aberrant ductus arteriosus. *Am. J. Dis. Child.*, Nov., 1931, *46*, 1148–1154.

Tracheo-esophageal fistula and atresia of the esophagus are not as rare as they are thought to be. The commonest type is a blind upper sac and a lower sac opening into the trachea above the bifurcation.

The clinical picture, the authors state, is clear cut. There is at first a constant flow of saliva from the mouth. When given food, the infant swallows once or twice without trouble but with every succeeding attempt the fluid is regurgitated through the mouth or nostrils. The child is then compelled to breathe with the nose, pharynx and mouth full of fluid. It suddenly stops nursing, its eyes become fixed, it rapidly turns blue, and then struggles to empty its lungs by coughing. It soon becomes deeply cyanotic, and these symptoms are repeated at each nursing. The stomach is slightly distended with air, while the rest of the abdomen appears flattened. For a few days the stools are normal in amount and are composed of typical meconium, then they become scant or absent. If present, they are the typical greenish-brown, slimy stools that characterize starvation. After the second or third day, there develops to a moderate degree the characteristic temperature of inanition. The infant rapidly becomes weaker, emaciated and more apathetic, and death takes place within two weeks. At autopsy practically all cases show a terminal bronchopneumonia.

The case here reported is one of atresia of the esophagus and tracheo-bronchial fistula in which a cause external to the anlage of the trachea and esophagus, but affecting its development, was apparent. The history was similar to the above; the roentgenograms, made after insertion of barium through a catheter showed that the upper portion of the esophagus

extended to the superior aperture of the thorax. The portion was dilated and filled with barium. Its lower end was smooth and rounded. No barium-filled tract extending from the esophagus to the trachea or penetrating into the adjacent structure was apparent. The entire bronchial tree from the larynx to the finer bronchi was visualized by the barium, and was apparently continuous with a large, dense mass of barium in the mouth, which was probably regurgitated from the upper part of the esophagus and subsequently aspirated. Further roentgenograms revealed conditions similar to the first findings, but in addition, extending downward from just below the left bronchus, there was a narrow fusiform shadow of barium, which reached to the cardiac end of the stomach. In the stomach, which was moderately distended with air, there were several dense shadows of masses of barium. No communication of the fusiform shadow and the lower end of the upper esophageal shadow or of the trachea or bronchi and this upper shadow was discernible. At autopsy, it was found that the esophagus consisted of two separate portions, proximal and distal. The atresia was brought about by a congenitally misplaced vessel (ductus arteriosus), which exerted external pressure

Jacobi and Rascoff review the theories of the causation of this type of anomaly. All are based upon the idea of an embryologic maldevelopment. They regard their case as corroborative evidence of Keith's view that external factors not inherent in the tracheal or esophageal anlage may be the determining ones.

They make the suggestion that intraperitoneal ligation of the cardiac end of the stomach, instead of intrathoracic ligation of the esophagus be combined with the gastrostomy usually performed in such cases.—*R. S. Bromer.*

Uspensky, A., and Helfon, A. Die Bedeutung der Röntgenstrahlen für die Erforschung der Bronchektasien. (The significance of the roentgen ray for the study of bronchiectasis.) *Fortschr. a. d. Geb. d. Röntgenstrahlen*, Nov., 1931, *44*, 635–648.

The authors discuss the roentgen visibility of bronchiectasis, using 13 brief case reports for illustration. The conclusion is reached that bronchiectasis may lead to various images according to whether there is secretion present within the bronchi or whether the bronchi are empty. Thus at times it appears in the form of ring shadows, at others in the form of a honeycomb, and again in others as a more or less diffuse clouding. The differentiation from pneumonia associated with diffuse abscess formation, from gangrene of the lung and occasionally from malignant tumors is exceedingly difficult. Fibrotic changes of the lungs may lead to secondary bronchiectasis. In such instances, if the process is far advanced, a fibrosis of the pleura with obscuration of the entire lung field and displacement of the mediastinal and heart shadow to the affected side may result. Bronchography is of value in demonstrating the morphologic appearance of the bronchiectasis as for example (*a*) saccular, (*b*) cylindric, (*c*) varicose, and (*d*) mixed type. If the bronchiectasis is localized chiefly to the left lower lobe the examination should be made with the patient in a horizontal position. Serial films showing the emptying of the bronchi are of advantage in estimating the motor function. The method further permits the demonstration of occult forms of bronchiectasis and the study of the tonus of the bronchi. The severe forms are not infrequently associated with generalized hyperplastic periostitis.—*T. Leucutia.*

Podkaminsky, N. A. Aspect radiologique de sidérose des poumons. (Roentgenologic aspect of pulmonary siderosis.) *Bull. et mém. Soc. de radiol. méd. de France*, June, 1931, *19*, 283–285.

The author has taken 140 roentgenograms of iron mine drillers to determine the extent of the pulmonary siderosis produced. It must be mentioned that the quartzite contained about 40 per cent Fe_2O_3 and about 60 per cent SiO_2, and that the mine contained about 93 per cent Fe_2O_3 and about 7 per cent SiO_2.

The following conclusions were reached: (1) The dust of iron mines leads to pneumoconiosis only after a lapse of a long period; (2) the pulmonary fibrosis produced by the siderosis and other pneumoconioses is due to the action of SiO_2, the injurious effect of the silicates being diminished by the protective property of the iron element; (3) there are three types of pulmonary siderosis: (*a*) the siderosis of metal polishers, the so-called *silico-siderosis gravis sive abrasiva*; (*b*) the siderosis of iron mine workers, that is, *silico-siderosis levis sive ferrariarum*, and (*c*) the siderosis of glass polishers, that is, *siderosis pura sive chromatica.*—*T. Leucutia.*

ACTION OF ROENTGEN RAYS AND RADIUM

ON THE

HEART AND LUNGS

EXPERIMENTAL DATA AND CLINICAL RADIOTHERAPY

BY

A. U. DESJARDINS

MAYO CLINIC

ROCHESTER, MINNESOTA

SECTION SEVEN

CONTENTS

(Current Number, October, 1932)

LUNGS

Clinical Considerations (*continued*) 131

(To be continued in the November, 1932, issue)

(Contents published in the January, February, March, July, August and September, 1932 issues)

PREFACE

HEART 1

General Considerations 1

Animal Experiments 1

Analysis of Experimental Data 44

Clinical Considerations 57

Conclusions 65

BLOOD PRESSURE

General Considerations 66

Animal Experiments 66

Analysis of Experimental Data 70

Clinical Considerations 71

Conclusions 72

SUPPLEMENTARY NOTE

Action of Roentgen Rays and Radium on the Eye and Ear . 73

RESPIRATORY TRACT—NOSE AND ACCESSORY SINUSES

General Considerations 75

Clinical Considerations 76

LARYNX

General Considerations 81

Animal Experiments 81

Analysis of Experimental Data 83

Clinical Considerations 83

LUNGS

General Considerations 93

Animal Experiments 93

Analysis of Experimental Data 115

Clinical Considerations 122

ACTION OF ROENTGEN RAYS AND RADIUM ON THE HEART AND LUNGS

Clinical Considerations (*Continued*)

Radiotherapy for Bronchial Asthma and Chronic Asthmatic Bronchitis. The term asthma has long been misused to designate a number of conditions featured by dyspnea; hence the expressions cardiac asthma, renal asthma, thymic asthma, and bronchial asthma. Cardiac and renal asthma have no place in the present discussion, because they obviously have nothing in common with true bronchial asthma. Thymic asthma refers to respiratory obstruction supposedly caused by pressure on the trachea by hyperplastic enlargement of the thymus gland. The influence of irradiation on the thymus gland and on thymic asthma will be dealt with later. Only the effect of roentgen and radium rays on bronchial asthma will be considered here.

It has long been thought that the crises of asthma are an expression of spasm of the bronchial muscles or turgescence of the bronchial mucosa induced by irritation of some portion of the respiratory tract by functional or organic disturbances, such as inflammatory deposits in the walls of the bronchi and certain chronic lesions of the nasal passages or mediastinum. It has also been recognized that the nervous system is more or less implicated; so much so, indeed, that some have regarded the disease as a variety of neurosis, in which spasm of the bronchial muscles or chronic hyperemia of the mucosa results from disturbed pneumogastric or vasomotor innervation. These views are now widely held, but the etiologic factors responsible for this distressing disease are still far from clear. The present tendency, however, is to restrict the diagnosis of asthma to those cases in which abnormal sensitiveness to proteins can be elicited. Every method of treatment which medical ingenuity could devise has been tried, but the very number of such methods indicates that none has been found preeminently satisfactory. A successful method will probably not be found until the nature and causes of the disease are better understood.

Having to deal with a patient with chronic asthmatic bronchitis and multiple nasal polyps, SCHILLING (1906) deemed it wise to exclude the possible presence of a foreign body in the bronchial tree by making a roentgenologic examination of the thorax. The following night, for the first time in three years, the patient did not expectorate, and the quantity of sputum remained much less than formerly. Astonished by this incident, Schilling then subjected to roentgen rays six other patients with chronic bronchitis, and in all six the quantity of sputum diminished greatly. Each of his patients was treated through two or more thoracic fields, each field being exposed to rays of about 12 Wehnelt for from 5 to $7\frac{1}{2}$ minutes. ECKSTEIN (1907) cited two cases, one of bronchial asthma and one of asthma-like crises and dyspnea arising several months after a crushing injury to the thorax. Improvement in the condition of both patients began immediately after the first exposure of the chest to roentgen rays. Stimulated by the report of Schilling, IMMELMANN (1907) recorded ten patients with chronic asthmatic bronchitis or bronchial asthma, in which surprisingly favorable results followed irradiation of the thorax for ten minutes through each of four fields (anterior, posterior, and two lateral fields). LEVY-DORN (1908) also reported many successes, even in patients who had suffered from asthma for

many years. His impression was that suggestion had much to do with the improvement because, while the patients were led to believe they were being irradiated, some were not actually exposed to the rays. In three cases, however, suggestion did not play any part; the condition of one patient improved considerably after irradiation.

GOTTSCHALK (1909) placed on record ten cases in, which the patients had suffered from bronchial asthma or chronic asthmatic bronchitis for a long time, and in four of which severe symptoms had failed to respond to any other kind of treatment. The anterior, posterior, and lateral aspects of the thorax were exposed to 5 X (Kienböck) of roentgen rays. Some of the patients were irradiated a second time (4 X to each of four fields) after an interval of four or six weeks. The crises diminished in frequency and disappeared. In all these cases Gottschalk had found the hilar nodes enlarged, and he inferred that the beneficial effect of irradiation was due to action of the rays on the lymphoid elements of these nodes. SCHILLING (1909) reported having treated fifty cases of chronic bronchitis, with or without symptoms of asthma. Many of the patients had been ill for from ten to fifteen years. About one-half the number derived substantial benefit, and some were regarded as cured. The results in children were thought to have been especially favorable. In one-fourth of the patients the treatment had apparently failed to influence the disease. The earliest cases were irradiated[173] once over the thorax only, but in later cases the treatment was repeated. SCHILLING (1910) cited a total of sixty cases treated in this manner, and claimed that 25 per cent had been cured and 50 per cent considerably improved. In the remainder he had found only transient improvement or none at all. He stated that bronchiectasis associated with enlarged hilar nodes can be cured. Chronic purulent bronchitis in a man aged fifty-three years was said by MENZER (1919) to have been greatly benefited by combined treatment with vaccine and roentgen irradiation; expectoration increased at first, but later diminished gradually and stopped. It is impossible to be certain, of course, whether vaccination or roentgen rays are to be given the credit. Twenty-four additional cases of bronchial asthma, treated by irradiation of the thorax were reported, by KLEWITZ (1922). Seventeen patients derived benefit, and five had no further attacks, but it is not clear how long such remission may have lasted; in nine patients the attacks recurred after a number of months, but the crises were less severe; three patients remained free from symptoms for a few weeks only, but subsequent crises were less distressing. The treatment had no apparent effect in seven cases, but five of these had been treated only once. Klewitz gave one-third of an erythema dose to each of three anterior and four posterior thoracic fields, and in some instances this was repeated two or three times at intervals of from two to four weeks. Later, KLEWITZ (1924) gave his results in eighty-five cases treated by this method. Twenty-six of these had been cured and the patients remained free from recurrence; twenty-seven were exempt from crises for several months, and recurring attacks almost always disappeared under additional treatment; in twelve others the crises disappeared for a few weeks only, and the remaining twenty patients did not derive any perceptible benefit.

MARUM (1924) added fresh evidence of the therapeutic value of roentgen irradiation in bronchial asthma. Of forty patients included in his report thirteen were classed as cured, eleven as greatly benefited, and eight as improved. The disease

[173] Exposed for from 20 to 300 minutes to unfiltered roentgen rays, the quality of which was given as 12 Wehnelt.

failed to respond in five others, and the remaining three had discontinued treatment and were therefore excluded. The hilus of each lung had been exposed, through two anterior and two posterior fields (total of four fields, each 80 sq. cm.), to roentgen rays with a penetration equivalent to 10 or 12 of the Wehnelt scale and filtered through 3 mm. of aluminium. The surface dose to each field was one-fifth of the erythema dose, and this was repeated two or three times at intervals of three weeks. In one patient the spleen had been irradiated without success. The immediate effect of irradiation was to increase expectoration, but this was followed by gradual diminution. Marum admitted that improvement in some cases might have been of the nature of a psychic reaction and did not offer any other explanation. One of his patients was well over six years, three for five years, two for four years, three for two years, six for from six to twelve months, and nine for a short time only. The following year KLEWITZ (1925) reported having treated a total of one hundred and thirty-one patients with bronchial asthma, always by exposing the thorax to roentgen rays. Of this number thirty-six were said to have been cured, forty-two greatly improved, twenty improved, and thirty-three remained unaffected. Equally favorable results were reported later by STERN (1925), BOLSCHAKOVA (1927), and TORREY (1927), and others testified to the therapeutic value of roentgen rays in asthma and chronic asthmatic bronchitis. KOTTMAIER (1928) stated that roentgen-ray treatment of asthma, chronic bronchitis, and bronchiectasis resulting from mediastinal lymphadenopathy exclusive of tuberculosis, had been effective in more than one hundred cases.

The favorable effect of irradiation in the foregoing reports was obtained by exposing the mediastinum and lungs to the direct action of the rays. Later, it was found that substantially the same effect could be produced by irradiating the region of the spleen. DREY and LOSSEN (1920) described the case of a patient with chronic lymphatic leukemia, enlarged spleen and mediastinal nodes, and supposed bronchial asthma, in which roentgen irradiation of the splenic, hepatic, inguinal, and axillary regions was followed by reduction in the size of the enlarged nodes at the root of the lungs and by corresponding abatement of the respiratory difficulty. Even though the patient was said to have been distressed by respiratory embarrassment for sixteen years, it seems probable that this was secondary to hyperplasia of mediastinal nodes and cannot be regarded as an example of true bronchial asthma. It is also possible that the shrinking of the nodes and the accompanying disappearance of dyspnea may have been a direct action of the radiation directed to the axillary regions and may not have had any relation to the radiation effect on the spleen, liver, and other structures exposed. The absence of a detailed description of the method used in irradiating the axillae leaves this point obscure. The inference of Drey and Lossen that the reduction in size of the hilar nodes and improved respiration may have resulted from the circulation of hormones liberated in the spleen by irradiation may seem far-fetched; but, as we shall see, others were impelled to test this idea in authentic or supposed cases of bronchial asthma. Thus GROEDEL (1921) reported having treated thirty cases by irradiating the region of the spleen, and only four patients did not obtain any improvement. The results were favorable in nine, very favorable in six, and brilliant in nine; in the two remaining patients the effect of treatment was unknown. In 1922 GROEDEL and LOSSEN published a report of their results in forty-one additional cases; eighteen patients derived marked bene-

fit, the condition of six patients improved considerably, that of six others improved slightly, three cases gave negative results, and the effect of splenic irradiation in the remaining eight patients was uncertain, because they failed to complete the treatment. Groedel and Lossen did not claim that the improvement noted in most of these cases was permanent, and they did not pretend that mitigation of symptoms in certain patients might not have been an effect of suggestion. They thought the rays stimulated the spleen to produce substances akin to antibodies, which prevented the attacks of asthma, or that irradiation removed certain factors which hindered the action of endogenous products acting as antibodies. They assumed that thoracic irradiation, as practised by others, was less effective and also acted through an indirect influence on the spleen. GALLINO and TERRADA (1924) gave their results in twenty-six of thirty-one cases which they had treated by splenic irradiation alone, except one in which the thorax also had been irradiated. Four of the patients had had asthma for twenty years or more, three for more than fifteen years, and five for more than eight years. The treatment had been given through one field 10 by 12 cm. corresponding to the region of the spleen, with roentgen rays generated at 200 peak kilovolts and filtered through 0.8 mm. of copper and 2 mm. of aluminium; one-half the erythema dose was given at a focal-skin distance of 30 cm. Irradiation was repeated twice at intervals of thirty days. Twelve patients had no further attacks; the crises diminished in frequency, duration, and severity in ten others; the treatment had no appreciable effect in two, and two others did not return after the first treatment. Two patients were still free from symptoms fourteen months later; two, twelve months later; four, nine months later, and four had been well from six to eight months. Gallino and Terrada did not accept Drey and Lossen's idea of hormones liberated by a spleen stimulated to increased function by exposure to the rays, but did not offer any other explanation. PÖHLMANN (1925) related having treated forty-five patients with bronchial asthma by irradiating only the region of the spleen, and claimed that all but four had derived great benefit. He thought the action of the rays was to raise the "stimulation threshold to antigen stimuli leading to anaphylaxis." EIMER (1925), who observed transient improvement in only one of four cases that had received one-third of an erythema dose through each of two anterior and two posterior thoracic fields over the root of each lung, obtained better results by directing the rays to the region of the spleen. Of nine patients treated in this manner two did not have any asthmatic crises for months, the condition of five others improved for a number of weeks, and the treatment was fruitless in two. Eimer thought the rays had an undoubted desensitizing action and also considered a possible influence on the suprarenal glands, but did not explain the nature of such action. WALDBOTT (1925) cited the case of a boy aged six and one-half years, who had suffered from asthma (dyspnea, cyanosis, wheezing)for four years. His submaxillary nodes were abnormally large, and roentgenologic examination of the thorax gave evidence of extensive, bilateral "infiltration" of the root of each lung) (probably enlarged nodes). Exposure of the splenic region to roentgen rays of medium wave-length was followed by complete cessation of the asthmatic crises, and the boy was still well eight months later. The action of the rays on the spleen was thought to stimulate the formation of antibodies, but the manner of such stimulation was not explained.

From the foregoing it would appear that irradiation of the splenic region is as

effective against asthma as exposure of the mediastinum and lungs. Others obtained similar results by combined exposure of the thorax and spleen. MONER (1925) irradiated both the lungs and spleen in twenty-three cases of asthma through two anterior and two posterior thoracic fields corresponding to the root of each lung and through one or two splenic fields, according to the size of the patient. Each field received 5 Holzknecht units of roentgen rays filtered through 4 mm. of aluminium at a focal-skin distance of 40 cm., and this was repeated one month later. All the patients had been ill for years and had tried all the remedies in vogue at the time. In five cases the effect of treatment was unknown. Of the remaining eighteen, seven were classed as completely cured, because the patients were still free from crises or other symptoms when the report was made three years to six months later. Marked improvement was noted in three patients; the treatment had no apparent effect in six patients, and two others were still under observation. RAMIREZ and COLE (1925) employed this combined method in thirty-five cases of bronchial asthma which had resisted all other treatment; many patients had enlarged tracheobronchial lymph nodes. Rays generated at 200 peak kilovolts, filtered through 0.75 mm. of copper, at a focal-skin distance of 50 cm., were used. In some patients only the thorax was exposed through four fields (anterior, posterior, and bilateral), while in others both the thorax and spleen were irradiated for from ten to twenty minutes. One patient, who had been a victim of asthma for ten years, was completely relieved for nearly one year by treatment of the thorax only; recurrence of symptoms yielded promptly to fresh irradiation of the chest. The condition of five other patients improved for more than six months, and five obtained only temporary benefit. RAULOT-LAPOINTE (1925) thought bronchial asthma was especially amenable to thoracic (mediastinal) irradiation, but that additional splenic irradiation was sometimes necessary. According to him, asthma of digestive derivation responded best when the liver was irradiated, and when the disease was related to endocrine disorder (thymus, hypophysis, thyroid) the corresponding gland should be irradiated. GERBER (1925), without giving the number of cases on which his opinion was based, also wrote favorably of combined irradiation of the thorax and spleen. He gave to each of three fields,[174] on three successive days, between 8 and 15 per cent of the erythema dose of rays of rather short wave-length filtered through 0.5 mm. of copper and 1 mm. of aluminium; the exposure was repeated after three or four weeks, if necessary. The results in many cases were expressed as excellent and in some instances as brilliant. Gerber also observed an increase in the quantity of sputum for a short time after treatment. MÜLLER (1925) reported a series of twenty-two cases treated by combined irradiation of the thorax and spleen. Twenty-seven per cent were classed as having been greatly benefited, 50 per cent as improved, and 4 per cent as unaffected; the records of the remainder were incomplete. One-third of an erythema dose was given to the hilus of each lung, from the front and back, and also to the spleen. Müller related the interesting observation of a woman with asthma and uterine fibromyoma, whose respiratory crises diminished greatly for about one year following a castration dose of roentgen rays to the uterus and ovaries. BEAUJARD and LEGOFF (1926) claimed to have obtained good results in six cases of infantile asthma by exposing the thorax and spleen to small doses through large fields. KOGAN-JASNY and ABRAMOVITCH (1926) treated eight patients with bronchial asthma by irradiat-

[174] Anterior and posterior fields over the mediastinum and one field over the spleen, each approximately 20 cm. sq.

ing the lungs, exclusive of the cardiac region, through seven fields according to the method of Klewitz; each field received from 20 to 30 per cent of an erythema dose of rays filtered through 0.5 mm. of zinc. In thirteen other patients the region of the spleen was exposed, through one field 12 by 12 cm. (Groedel method), to 25 per cent of an erythema dose of rays filtered through 4 mm. of aluminium. In a third series of fourteen patients both the lungs and spleen were exposed through several fields, each of which received 20 per cent of an erythema dose. This was repeated after three or four weeks if the first course of exposures remained without effect. Of the thirty-five patients nine were considerably benefited, the condition of twenty-three improved, and the physical situation of the three remaining patients was not modified. Kogan-Jasny and Abramovitch expressed the opinion that combined irradiation of the lungs and spleen yielded the best results. In many cases they also noted an early change in symptoms, such as increase in expectoration and improvement in respiration.

Strelkov (1927) had twenty-three cases of severe asthma, in ten of which evidence of enlarged tracheobronchial lymph nodes was found. The patients had been ill from two to fifteen years and had tried every possible form of internal medication and other methods of treatment, without success. Twenty per cent of an erythema dose was given to the root of each lung and to the spleen (two anterior and two posterior thoracic fields, each 100 sq. cm., excluding the cardiac region, and one anterior field over the spleen). In no case was the disease cured, but the condition of four patients improved greatly, and they were free from symptoms for about five years; that of eight patients improved considerably, and the crises and other symptoms disappeared for two or three years. In nine patients improvement was rated as slight, and the condition of two other patients remained unchanged. Strelkov also thought combined irradiation of thorax and spleen more effective than either alone. Of the many contributions on the subject one of the most interesting is that of Valléry-Radot (1927). Thirty-one cases of asthma, eight cases of hay fever, and twenty-five cases of asthma associated with hay fever were treated with roentgen rays. About half the patients were males, and nearly all were adults; in many cases the disease was of long standing and had resisted different kinds of treatment. In some cases irradiation was directed to the hilus of the lungs (mediastinum) through an anterior and a posterior field; in other cases the region of the spleen was irradiated through an anterior and a posterior field, while in still other cases, both the thorax and spleen were exposed to rays of medium wave-length, filtered through from 5 to 10 mm. of aluminium, for ten minutes,[175] ten or twelve times at the rate of two sessions a week. The crises of asthma or coryza, or both, disappeared completely in nineteen cases, and some of the patients were still well twelve, fifteen, and even eighteen months after the completion of treatment. Substantial and lasting improvement occurred in sixteen cases, and the treatment was practically ineffective in twenty-nine other cases. In some instances, irradiation had a truly remarkable influence. One patient, who had had violent crises of asthma day and night for twenty-five years was completely relieved after the fourth irradiation and was still well fifteen months later. Valléry-Radot had the impression that combined irradiation of thorax and spleen had the greatest effect, and, in many cases, improvement did not begin until the patient had been irradiated six, eight or even ten times.

[175] 500 R (Solomon) per field, per session.

If we delve further into the subject we find HAJOS (1923) assuming that roentgen rays desensitize the body to protein and obtaining equally favorably results in three of seven patients with bronchial asthma by exposing the thorax and hilar nodes, liver, and spleen, to from one-third to one-half the erythema dose of roentgen rays. Hajos correctly ascribed asthma to increased sensitization to proteins, but claimed that the apparent desensitization brought about by irradiation was due to transient lesions of the liver caused by the rays. PRITZEL (1923) thought asthma was the result of disturbances of function of the thyroid and suprarenal glands. He exposed alternately the upper part of the lungs and thyroid and the lower part of the lungs and suprarenal to from one-sixth to one-fourth of an erythema dose. He thus treated forty cases of asthma, thirty-five of which remained free from crises for a short or long time, and regarded this effect as due to direct or indirect action of the rays on the nervous components of these glands of internal secretion. WIDAL and ABRAMI (1924) gave a detailed report of four patients apparently suffering from both hyperthyroidism and asthma, without pulmonary lesions. Many other kinds of treatment had been tried previously without effect, but irradiation of the thyroid gland was followed by recovery from the thyroid disturbance and by disappearance or marked diminution of asthmatic manifestations. NESTEROV (1926) held that irradiation of the tracheobronchial lymph nodes can influence the symptoms of asthma only so far as nerve roots and nerve endings are struck by the rays. He declared having obtained superior results by irradiating also the cervical ganglia, medulla, and cerebral hemispheres, and claimed permanent cure in over 36 per cent, improvement in over 45 per cent, and failure in over 18 per cent of his cases, the number of which was not given. JOUGUENBOURG (1927) described two cases of asthma complicated by hyperthyroidism, in which remarkable improvement followed exposure to roentgen rays of the region of the thymus and of each lobe of the thyroid; and the favorable influence of the rays was attributed to their action on the thyroid gland. Assuming that asthma is due to disturbed equilibrium of the vegetative nervous system, PODKAMINSKI (1927) exposed the occipito-cervical region to 25 per cent of the erythema dose of roentgen rays[176] filtered through 4 mm. of aluminium. Eight patients treated in this manner were observed for from two to nine months. All were said to have benefited, and five had no further asthmatic crises. No attempt was made to explain such effect.

On the assumption of an ill-defined relation between the hypophysis and bronchial asthma, ASCOLI and FAGIUOLI (1920) claimed, and submitted the records of five cases to prove, that repeated exposure of the hypophysis to small doses of roentgen rays had favorably influenced the patients' symptoms by stimulating this much maligned gland. The condition of the patients was said to have improved substantially, but the duration of improvement was not mentioned. But, since the patients also received pituitrin or adrenalin when the effect of irradiation seemed insufficient, the ensuing relief may have resulted from the medication rather than from the action of roentgen rays. EPIFANIO (1921) described two additional cases of bronchial asthma, in which irradiation of the hypophysis, through a frontal and two temporal fields, abated the crises in one and stopped them completely in the other. Favorable results were also obtained in a patient with cardiac asthma, but no details were given, except that the hypophysis was exposed to the rays three or four times at

[176] Quality corresponding to 7 of the Bauer scale.

intervals of a week or ten days. KAUM (1926) also claimed to have improved the condition of five patients by irradiating the hypophysis through the mouth.

Still more interesting perhaps was the communication of SCOTT (1926), whose attention to the subject was first attracted by the undoubted influence of roentgen rays on chronic bronchitis in many patients receiving general irradiation[177] for carcinoma of the breast. The extreme gratefulness of one patient, who said that the asthma which had caused her great distress for fifteen years had disappeared completely, led Scott to ascertain the therapeutic value of roentgen rays in this disease. He found that the patients derived benefit within a few hours; expectoration increased, the sensation of tightness of the chest diminished, and doses of 10 X (Kienböck) of rays generated at low voltage were sufficient. General irradiation (entire trunk) once or twice a week, with an interval of two weeks after the fourth dose, and then once a month for four months, gave better results than exposure of the thorax only. Scott noted that patients exposed to the strong dose of 20 X developed acute bronchitis, and, because this did not occur after small doses, he concluded that bronchitis may have been an effect of the rays. Twenty-one patients, who had previously tried every other known treatment, obtained some measure of relief, except one who had marked emphysema.

The patients included in most of the preceding reports suffered either from true bronchial asthma or from chronic asthmatic bronchitis, but it is probable that the respiratory disturbance in a certain number was due to mechanical pressure on the vagus or its branches by enlarged mediastinal lymph nodes. Until recent years a distinction between true bronchial asthma and other pulmonary disorders characterized by recurring attacks of dyspnea was seldom made, and many cases of so-called asthma really should have been classed as leukemia, lymphoblastoma, thymic hyperplasia, etc. The tendency now is to designate as bronchial asthma only those cases in which a definite protein reaction can be obtained, but this criterion may not be absolutely sound, because of the probability that different etiologic factors may give rise to the same pulmonary reaction. At least such is the natural deduction to be made from the valuable experiments of BRODIE and DIXON. Regardless of the validity of this criterion, however, it is clear that dyspnea, cyanosis, and other symptoms suggestive of asthma but related to diseases of which hyperplasia of the mediastinal and bronchial lymph nodes is a common feature, should be considered separately. A good example of this is the case reported by Drey and Lossen.

Even if a generous proportion of the cases reported as having been cured or benefited by roentgen-ray treatment be excluded for inaccurate diagnosis or as probable examples of the common influence of suggestion, there still remains an imposing number in which the effect of irradiation can hardly be gainsaid. But when the mechanism of such action is considered, the first impression is that suggestion must have played an important part. The most salient features are that, while the results obtained by different workers have varied to some extent, the average of cases cured or greatly improved is over one-half of the total, and that approximately the same degree of benefit has been observed after irradiation of the mediastinal nodes and lungs, spleen, thorax and spleen, or even of other regions, such as the neck and intracranial structures. It is essential to bear in mind, however, that while certain authors

[177] Scott believed that general exposure of the trunk (front, back, and sides) to roentgen rays was the most effective method of treating cancer.

stressed the action of the rays on the thyroid or suprarenal gland (Pritzel, Jouguenbourg), on the hypophysis (Ascoli and Fagiuoli, Epifanio, and Kaum), on the liver (Hajos), on the brain, medulla, and cervical ganglia (Nesterov, Podkaminski), or on the body as a whole (Scott), and the structures mentioned were undoubtedly exposed to the rays, the major and essential effect may have concerned other structures in the same region. There is little ground for the belief that therapeutic doses of roentgen or radium rays exert any perceptible influence on nerve cells and nerve tissue. On the contrary, it has been established that the specific elements of the nervous system are exceptionally resistant to irradiation. Hence, any effect on bronchial asthma or chronic asthmatic bronchitis noted after irradiating the head or neck probably is not related to any action of the rays on the specific elements of the brain, spinal cord, vagus, or on the ganglia of the sympathetic system. The significance of this deduction appears the greater if we remember that, in many of the foregoing reports and regardless of the organ or region treated, the symptoms abated after one or two exposures to from one-fourth to one-half of an erythema dose of roentgen rays of medium or short wave-length. Under such conditions, it is not likely that the rays could have yielded therapeutic results through some doubtful effect on glands of internal secretion, such as the hypophysis, thyroid, and suprarenal.

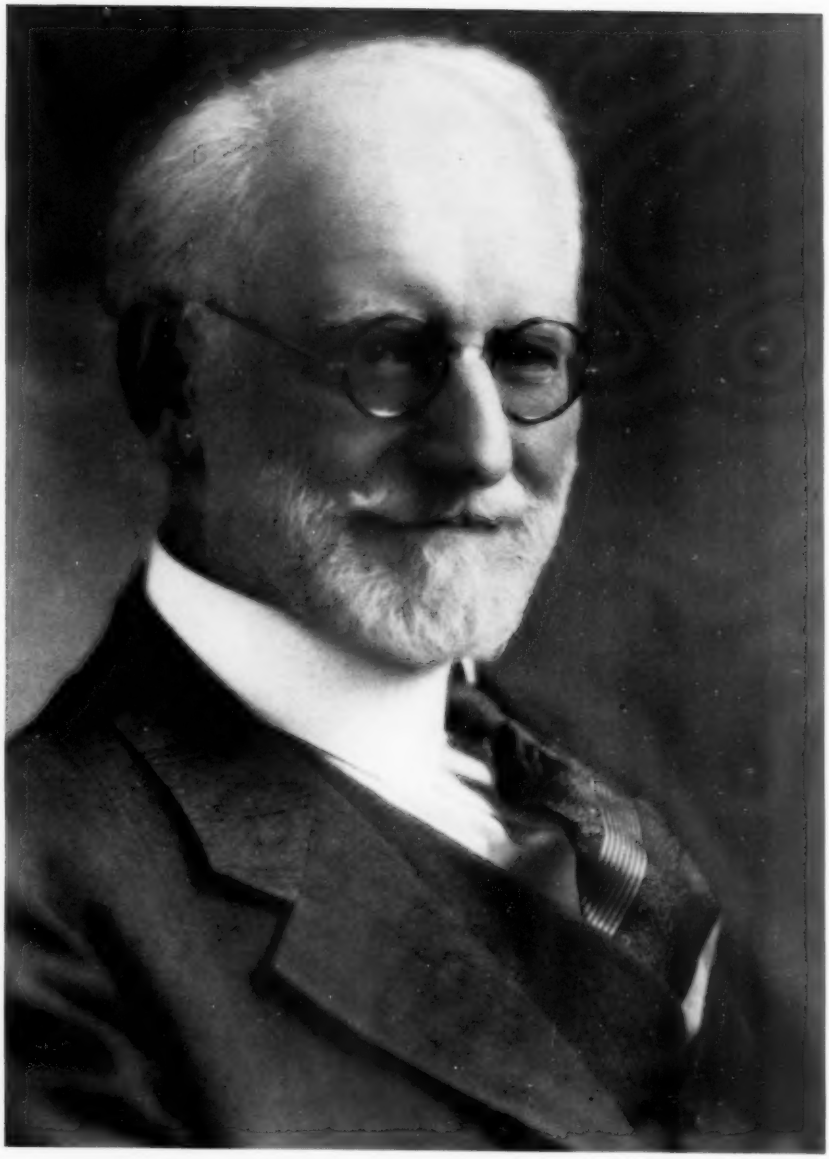
How, then, may the results be accounted for? It must be admitted at the outset that any explanation advanced must be largely hypothetical and may not take into account certain obscure factors. Nevertheless, material for a reasonably adequate explanation is not wholly lacking. Attention must first be directed to the fact that the divergence between the interpretations advanced by different authors may be more apparent than real. Thus, the action of roentgen rays on the tracheobronchial or pulmonary lymph nodes assumed by Gottschalk, Schilling, Klewitz, Eckstein, and Bolschakova, the circulation of hormones mentioned by Drey and Lossen, the elevation of the stimulation threshold to antigen stimuli leading to anaphylaxis advanced by Pöhlmann, the stimulation of antibodies suggested by Waldbott, the desensitization of the body to proteins offered by Hajos and by Eimer, the diminished hyperesthesia of Gerber, and the complex indirect influence on the whole body proposed by Scott may all be only different ways of expressing much the same idea or, at least, may possess a common factor, whether the rays were directed to the mediastinal nodes and lungs only, to the spleen only, to both the lungs and spleen, or to other regions. Many authors have written that they irradiated the hilus of the lungs, the tracheobronchial lymph nodes, the lungs themselves, the spleen, the thymus, thyroid or hypophyseal glands of internal secretion, or still other structures, and have attributed improvement in the condition of patients to action of the rays on the organs or structures mentioned. But whether the aim of the radiologist is to irradiate the hilus of the lungs, the tracheobronchial lymph nodes, the thymus gland, or the lungs themselves, it must be apparent that a large volume of blood in the great vessels, lungs and heart is exposed to the rays. When the aim is to irradiate the thyroid gland or hypophysis it must be obvious that a considerable mass of blood in the thyroid or brain is exposed to the rays. Also, the fact that exposure of so many different structures should yield essentially similar results constitutes strong presumptive evidence of a common factor.

What may this common factor be? Probably the well-established and exceptional

radiosensitiveness of leucocytes, especially the lymphocytes, in the spleen, lymph nodes, circulating blood, lungs, liver, thymus, and other organs. Exposure of lymphoid organs and tissues or of the circulating blood to moderate doses of radiation is known to cause rapid and extensive destruction of leucocytes, but especially of lymphocytes. The relation of such destruction to the influence of the rays on many physiologic and pathologic processes appears much more significant than is generally thought. Moreover, in connection with the problem of the action of radiation in asthma, Müller (1927 and 1928) has furnished pertinent information. He exposed the root of the lungs of asthmatic patients and also of non-asthmatic persons (controls), thirty subjects in all, to one-third of an erythema dose of roentgen rays, and studied their metabolism with reference to urea, uric acid, residual nitrogen, blood viscosity, refraction, and also the surface tension of the serum. He found in both groups a slight increase in urea, a tendency of the serum protein to decrease, and a diminution of leucocytes, especially lymphocytes. When the condition of the patients improved, the number of eosinophiles diminished, the blood calcium increased, and the potassium decreased. These findings led him to conclude that irradiation reduced the anaphylactic components, decreased the potassium and vagotonus, and that these alterations were accompanied by a compensating increase in calcium. If the assumption that lymphocytes and other leucocytes represent the first line of defense of the organism against many noxious influences is warranted, the corollary that these cells must contain or elaborate within themselves the protective substances which give them their defensive power must also be accepted. It follows that the destruction of a sufficient number of such cells must liberate the protective substances into the blood stream and thus make them more readily available for defense. In other words, irradiation can be said to induce a sort of autogenous protein reaction which may be quite as effective as the reaction caused by protein substances injected into the patient. In the case of asthma, some such mechanism undoubtedly operates and acts to desensitize the body to proteins or to release the vagus and its pulmonary branches from the abnormal pressure of hyperplastic lymphoid structures (mediastinal lymph nodes or thymus gland). Based on sound and adequate experimental premises, this hypothesis has the additional merit of reconciling the many different views which have been expressed heretofore and of providing a rational explanation of the influence of irradiation on asthma.

Another possible element of the problem must be borne in mind. A common clinical feature of asthma is increase in the number of eosinophiles in the blood. Recent studies indicate that such eosinophilia has an important bearing on the course of the disease. Eosinophiles are not so radiosensitive as lymphocytes; their susceptibility to irradiation is approximately the same as, or perhaps slightly less than, that of polymorphonuclear leucocytes. No doubt this factor also must be taken into consideration, but until the significance of eosinophilia in asthma and its relation to the disease have been determined, the exact influence of irradiation cannot be absolutely appraised.

To be Continued.



AUGUSTUS W. CRANE
Caldwell Lecturer, 1932